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RICHMOND STEPHENS, M. D.

VOLUME IV^{2} *NUMBER 4*

THE CLINICS

OF

JOHN B. MURPHY, M.D.

AT

MERCY HOSPITAL, CHICAGO

AUGUST, 1915
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RICHMOND STEPHENS, M. D.

Volume IV THE CLINICS OF JOHN B. MURPHY* Number 4

TALK ON SYPHILIS

Summary: Early diagnosis and early treatment; sodium cacodylate *vs.* salvarsan; frank course of preputial chancre; occult course of syphilis when chancre is in urethra; invasion of blood- *vs.* lymph-vessels—anatomic reasons; mode of administration of sodium cacodylate; prevention of contagion of syphilis.

Dr. Murphy (October 8, 1914): We have two very interesting cases that came into the office yesterday. The first is that of an engineer who contracted a chancre of the tongue, at its middle, from smoking a pipe that had been used by his syphilitic fireman. At the time of his first visit, eight days previously, we immediately scraped his tongue, excised a piece, and stained for spirochetes, the detection of which confirmed the diagnosis. Then we instituted what we believe to be the best method of treating early syphilis, namely, daily hypodermic injections of sodium cacodylate. I recently recommended salvarsan, but I have returned to my first love, which I originally suggested and used before we had "606." Upon his return yesterday the chancre had shrunken to one-sixth of its original size. I know that it will be healed when he returns for the next visit, six days hence, allowing just two weeks from the time of the original sodium cacodylate injection to that of complete healing. Usually chancres heal within from six to seven days, and much faster with sodium cacodylate than with salvarsan. The speed of repair is accelerated by putting chrysolate of argyrol over the surface of the sore at the first visit. Owing to the great increase in the price of salvarsan, from $30.00 to $35.00 an ounce, I think sodium cacodylate is the therapeutic agent of the future. Fifteen cents' worth is sufficient to cure a chancre.

We are very much impressed with the rapidity with which the primary lesion is healed by sodium cacodylate. As to the permanency of the cure, we are not so certain, but we do know that "606" has been a failure as regards permanency of cure and a great disappointment.

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* This number of the Clinics was edited by Dr. P. G. Skillern, Jr., Philadelphia.

The second case was that of a doctor who, despite an abrasion on the back of his finger, examined a patient with ungloved hand and contracted a typical chancre. He will likewise be treated with sodium cacodylate, beginning with two grains hypodermically. Unfortunately, his disease is in a much later stage, because the eruption is just commencing to appear. The drug will be rapidly increased up to five grains a day in an endeavor to prevent involvement of the central nervous system.

There are some peculiarities about the locality of the primary lesion in these syphilitic infections that lead to very serious sequelæ. When the chancre involves the prepuce, the central nervous system is invaded much later than when the chancre is situated in the rich vascular tissue of the urethra. For instance, if in the presence of gonorrhea a chancre develops in the urethra, the syphilitic infection is by far more liable to involve the central nervous system early. So common is this that we never analyze a case of this class with a history of gonorrhea without exacting all the details of the attack, if there is anything in the patient's make-up that arouses a suspicion of syphilis, because when the chancre locates in the urethra, the spirochetes never of themselves give rise to the pronounced lymphatic infection and the adenopathies. Furthermore, the cutaneous eruption is conspicuous by its absence. Patients with the urethral portal of entry never have alopecia as a secondary sign. Why? Because with a specific infection of the urethra the spirochetes enter the bloodstream directly, and *not* through the lymphatics to the bloodstream. The anatomic reason is that the urethra has no submucosa: it has a basement membrane, but no submucosa. It has just a vascular network on the corpus spongiosum, so that the spirochetes enter the blood directly, and the syphilitic infection first manifests itself by a central nervous system lesion. For example, we had a man here the other day from Albuquerque, New Mexico, who positively denied any infection, but who had a urethral discharge which had been prolonged, but at no time profuse. Get the idea!— *prolonged, but not profuse:* it was the urethral spirochetic infection that caused the slight discharge and minimal irritation, which continued for a long time. He came in with a cerebral thrombosis developing after an epileptic attack. He gave a quadruple plus Wassermann, without anything at all suggestive in the history. Think of it! This emphasizes the clinical course of primary urethral chancre infections.

Visiting Doctor: How long after the appearance of the initial lesion do you give the sodium cacodylate?

Dr. Murphy: I give the sodium cacodylate immediately after the appearance of the initial lesion, and keep it up until the external manifestations have entirely disappeared—two weeks, three weeks, four weeks. First we commence with two grains once a week, always starting with the smaller doses. Why? Because some patients have an idiosyncrasy to arsenic. This has been known as long as arsenic has been known. If the idiosyncrasy is marked, it gives the patient the garlic breath. If there is no idiosyncrasy, the drug is pushed up to five grains. The sodium cacodylate is marketed in ampules. We never mix it ourselves, and never let the druggist mix it. We have not given it intravenously: perhaps that will be done. Nothing is more beautiful than the healing of a chancre by sodium cacodylate. We used it before we had "606." In one of my original articles I cited the case of a girl who had been infected from a drinking-cup, developing a chancre on the lip, which, under sodium cacodylate, healed completely in eleven days. If we can cure the primary lesion in six, eight, or ten days, the syphilis problem is mastered. Why? Because the danger of transmission is thereby minimized. It is from the primary lesion and the secondary manifestations and discharges that contagions occur. We can have the chancre patients arrested and detained in the hospital for a week or ten days, but they cannot be forced to quit business for twelve weeks or more, as was necessary for healing in the old treatment. That is the way the problem is going to be solved. The question as to whether sodium cacodylate permanently cures each individual is not so paramount as the knowledge that it is the factor that puts him in dry-dock while the danger of transmission is active. It is the healing of the primary sore that stops the transmission and that can be done very readily.

TUMOR OF THE PAROTID SALIVARY GLAND

Summary: Clinical history of illustrative case; surgical pathology of tumors that occur in or near the parotid gland; case showing malignancy of irritated moles; importance of preserving the facial nerve by careful dissection and hugging the tumor; electric response of facial nerve at operation; result of operation.

HISTORY

The patient, a female aged sixty-two years, married, the mother of nine children, was admitted to the hospital December 15, 1914. She had the usual diseases of childhood. In 1892 she had typhoid fever of the ambulatory type; duration, eleven weeks. Family history is negative. Menopause occurred in 1900.

The present trouble began two years ago, in October, 1912, the day after the patient had attempted to wear an ear-ring. A slight swelling appeared at the junction of the right ear and cheek: to this she paid no attention. By April, 1913, six months after the appearance of the swelling, the mass had grown to the size of a walnut, and she then consulted a physician, who poulticed the tumor and gave her medicine to be taken internally. Four months later, in August, 1913, she visited another doctor, who lanced the tumor, withdrawing a dark, bloody fluid. The wound discharged for two weeks and then healed. Five months later, in January, 1914, the tumor recurred, and she again consulted the first physician, who repeated his treatment of poulticing the tumor and administering medicine internally. The tumor was aspirated on December 13, 1913, but the patient does not know the nature of the fluid that was withdrawn.

COMMENTS AND OPERATION

Dr. Murphy (December 17, 1914): What is the classification of the tumors occurring in the neighborhood of the parotid gland?

Intern: First, tumors of the parotid salivary gland.

Dr. Murphy: Number one and most commonly. Number two is what?

Intern: Tumors of the mandible.

Dr. Murphy: Next in frequency to the tumors of the jaw is what?

Intern: Dermoids.

Dr. Murphy: Yes! That is one of the positions where dermoids occur. In the development of the branchial clefts a part of the cutaneous tissue remains submerged and encapsulated, and a dermoid arises. Next to the dermoids that appear at the canthus of the eye, dermoids are more frequently met with here than elsewhere in the body excepting the abdomen. They are involution tumors. That is one of the common types of tumor in this position. The *most* common, however, are the tumors of the parotid salivary gland. How do you classify them?

Intern: As malignant and non-malignant.

Dr. Murphy: The parotid salivary gland has a greater variety of tumors than any other structure in the body excepting the central nervous system. The so-called mixed tumors are very common in the parotid gland—mixed as concerns the mesoblastic elements, consisting of chondroma, fibroma, osteoma, cysts, and dermoids. Not only these, but you have also mixed cells in which it is impossible for the pathologist to distinguish them from cells of the epithelial type. The prevailing cell is an endothelial or mesothelial cell. By many these tumors are considered as varieties of carcinoma. By others they are called endotheliomata, so that there exists a great difference of opinion about just which class these tumors belong in, and in some tumors there are often found many varieties. That is why they are so complex.

Now, the question arises in this case, what is its origin? Did it begin above or below the facial nerve? My impression is that it is anterior to the nerve, and that the safest incision is to cut right through in that direction on the capsule, as one cannot tell in which direction the facial nerve is displaced here; and then hug the tumor closely. The danger of the operation has been clearly explained to the patient: this danger is the injury to the facial nerve.

The gross appearance of the tumor is no criterion as to its variety. The fact that it comes out in that way does not signify that it is mesoblastic nor that it is epithelial. We must endeavor to get away the sac of the tumor. You see that cystic fluid is merely a secondary affair, and not a primary one. Inspection shows that it is a solid tumor that has broken down. That is the rule with these cysts. I want to get on the capsule of the tumor and then do a spreading dissection—that is safest for the nerve. Usually these tumors are fairly well encapsulated, because of their mesothelial and endothelial lining. The epithelial lining means what? That the entire capsule must be removed. It is really a matter of grubbing, and grubbing with infinite care, keeping as close to the connective tissue of the sac as is possible. Sometimes these are very wicked tumors, but the carcinomatous tumors in this position are not nearly so wicked in the rapidity of destruction as are the sarcomata. The sarcomata can cause a fatal termination in a very short period of time. When a sarcoma begins in the skin or in the parotid gland itself,—occasionally it starts in the derma and then passes down into the parotid,—it is rapidly fatal. We had a very striking case of that in my late associate, Dr. Lemke. He had a small mole on the cheek. He was toying with it one day when he had nothing else to do. Thinking it contained some material, he pressed it and pinched it. It began to enlarge. He was in California at the time, and he underwent a fairly radical operation, a square inch or more of its structure having been extirpated. Again it began to enlarge. He came to Chicago about six weeks after the operation, and upon examining it we found that it had penetrated into the parotid gland. In removing it we made a very extensive dissection. He returned to California, and in another six weeks was dead from metastases in his lungs, showing what a very virulent and wicked type of tumor it was.

Now we are back to the normal gland again. There is the capsule of the tumor. I am excising the upper portion of the tumor and sending it to the pathologist for an immediate report.

This growth is extirpated, working on the basis that it is mesothelial lined. Now we take off a larger section, and you see the depth of it: this demonstrates how deceptive these tumors are.

Let the record show that, notwithstanding the extensive removal, we have hugged very closely the wall of the sac in order to avoid the facial nerve; and also that, in placing the electrode beneath the ear on the distal portion of the wound, all the muscles of the face are seen to contract.

Had I cut the nerve, its distal end would still respond to the electrode because the axons are not degenerated until about eighteen hours after section. Upon testing the proximal end of the nerve behind the tumor I obtain a full and clear response, showing that we have succeeded in displacing the nerve intact. That is a source of great satisfaction. Here is the normal parotid, which you recognize at once. There is the granulation tissue characteristic of this neoplasm. We have a good conformation here. A mattress stitch gives the best approximation in this situation.

Let the record show that the tumor was enucleated close to its wall, and, we believe, with a complete removal; that it did not invade the neighboring structures; that it was firmly adherent, as these tumors commonly are, even when they are not malignant; and that a strand of silkworm-gut was inserted for drainage, to be removed at the end of forty-eight hours. If any trace of the capsule of one of these tumors is left behind, the tumor recurs.

It is needless for me to add that the putting in of the ear-rings had nothing to do with this malady.

Let the record show that, as estimated, the tumor originated and emerged below the trunk of the facial nerve; that the facial nerve rested above the neck of the tumor, and that we believe we have spared the nerve, since at the close of the operation it responded to the faradic current on the distal side of the incision.

The other salivary glands do not exhibit this type of tumor, since it is practically never seen in the submaxillary or sublingual glands, nor in the abdominal salivary gland, namely, the pancreas. Tumors in the pancreas exhibit great variety, but they are extremely rare. Tumors of the salivary glands are believed to originate from the endothelial cells lining the lymph-spaces; of course, this refers only to those of the mesothelial type, since true carcinomata are always epithelial; but the differentiation either clinically or microscopically is difficult. The enlargement of the parotid, with or without other salivary and lacrimal glands, described by Mikulicz in 1888, might complicate the problem of diagnosis.

The patient made an uneventful recovery. There was no paralysis of the facial nerve. She was seen at the office on February 17, 1915, two months after the operation, and there was no evidence of return of the tumor.

PLASTIC OPERATION ON SOFT PARTS OF NOSE

Summary: Clinical history of patient; preparation of the nose for a pedicled flap; preparation of a flap from the arm; necessity of a redundancy of flap-tissue; advantage of free blood-supply to face in viability of flap; apparatus to insure fixation of head to arm; exposure *vs.* dressing of wound; hyperesthesia of flap after operation.

HISTORY

The patient, a male aged forty-one years, married, the father of two children, was admitted to the hospital January 11, 1915, because of a deformity of the nares and difficulty in breathing.

His family and past histories are negative. He denies venereal infection. Seven years previously, in March, 1908, the patient's nose was frost-bitten, but healed within a few days, leaving a little pimple at the tip. About ten days after the nose was frost-bitten he scratched it, and an inflammatory process immediately began. There were no chills or fever and no discharge of pus. In order to abate the inflammation a poke plaster was applied, which relieved the pain immediately, but caused gangrene to appear twenty-four hours later. The face became more inflamed, and the swelling and edema were so great that the eyes were closed. He was taken to a hospital, and hot dressings were applied. In June, 1908, he was operated upon. After the operation the right ala was fissured, the left nostril obstructed, and the septum perforated.

COMMENTS AND OPERATION

Dr. Murphy (January 13, 1915): This is a nasty case. You see what has happened! He is a doctor. We must take the graft from the forearm or upper arm. You remember the flap we made about two weeks ago? It all lived, and we have not separated the last section yet, but intend doing that this morning. It was a very large flap, and was made in order to save a leg. We are packing the posterior nares to keep the blood from the pharynx when he breathes through the mouth. I am anchoring this edge with a suture so as to prevent its escape during the process of repair of the other flap. I must make a lining for the flap and free it as far as the bone. The nostril is destroyed down to its periosteum. We have the flap open all the way, and shall turn it down. That makes the underlying flap. The lining is really more essential than the outer flap. This step was necessary to prevent stenosis of the opening. That makes the entrance into the nose. This crushing forceps will stop all bleeding within a few seconds.

We employ rectal anesthesia, as you see. It has been very efficient, but now the patient is commencing to awake. I shall not disturb him until he is fully under again. That is a very good method of anesthesia, but we are a little slow in adopting it, since its preparation requires more time than the inhalation anesthesia.

I am suturing so extensively in order to tack the flap down firmly, and this is where the vitality of the flap will be taxed. It looks like a very large flap, but these flaps contract so greatly after they are sutured that, unless one makes a very large flap, he does not have one large enough at the completion of the work. We are using silkworm-gut.

Now we shall prepare the arm. By the time this work is completed all the bleeding from the nose will have stopped. See! the flap from the arm comes over the entire area without a particle of trouble. We put tacking stitches in between. If this entire flap lives, there will be plenty of skin,—even a redundancy,—so that one may fashion any kind of a nose. The flap seems rather long, but by the time we detach it and double it back upon itself, it will not be too long. The circulation should rapidly reëstablish itself, because of the very rich supply from the face, while the circulation in the arm itself is fairly good—much better than in the thigh. We are covering all abraded surfaces with bismuth (iodid) powder. The nurse will have a box of this powder, and every time she sees the wound oozing, she will dust some on. While the position is very irksome to the patient, yet it will have to be maintained for two weeks and possibly a little longer.

Let the record show that before the operation a plaster-of-Paris mask was made on the head and shoulder, so that the head and shoulders were immobilized; that when the flap was fixed to the nose, the hand was fixed to the mask with adhesive plaster, to prevent motion; that we exposed the nasal bone and elevated both flaps well out laterally; that we split both alæ, and then sutured the flap in first by catgut sutures to the face, to prevent formation of a hematoma, and then accurately sutured the flap *in situ*.

Dr. Murphy (January 26, 1915): To-day we detach the flap from the arm as you see. There is abundant nose now—in fact, a superfluous amount of nose. It seems to me that contraction is inevitable, so that all the superfluous tissue is needed. We sponge the arm where the flap was taken with 5 per cent. phenol because this area was exposed the entire time: despite this he has had no suppuration. The operative field has been constantly exposed to his breath, but such wounds do very much better exposed to the air than when covered up.

Dr. Murphy (February 5, 1915): In cutting the flap it was extremely sensitive, showing that the nerves of sensation have regenerated themselves, in consonance with previous experiments that grafts form the most sensitive portion of the skin.

[Note.—The patient had a beautiful result. The entire flap lived.—Ed.]

PLASTIC OPERATION ON SKELETON OF NOSE

Summary: Clinical history of patient; comments and operation; importance of avoiding injury to nasal mucosa; bone-graft from tibia modeled to form new bridge; importance of contacting graft with its bony bed; futility of paraffin; graft as osteoconductor.

HISTORY

The patient, a male, single, aged twenty years, was admitted to the hospital on February 10, 1915. His family history is negative; he had measles at the age of seven years.

At the age of eight years his nostrils became sore and discharged light yellow pus. A crust formed, and bled when picked by the patient. He was taken to a doctor, who prescribed sprays and irrigation for three months. At the end of this period of treatment, as the patient remembers, the secretion stopped. Then the bridge of the nose began gradually to sink in. At the close of one year the depression had reached the same depth as exists at present. No sequestrum appeared at any time. Two years ago the nose again became sore and bled, and there was a slight discharge of pus. Under treatment this stopped in about three weeks. As a rule, the patient can breathe through the nose without much difficulty, except during an attack of coryza. At night he breathes through the mouth.

COMMENTS AND OPERATION

Dr. Murphy (February 11, 1915): You will note that this is a very flat nose (Fig. 163). The lower portion of the nasal bone and septum and a portion of the nasal process of the superior maxilla have been destroyed. The cartilage in the tip is not destroyed. This has permitted the nose to sink in to a superlative degree, but the conformation of the tip and alæ is maintained, though their relationship to the face is changed, as the nasal openings look forward in place of downward and forward. The reconstruction of the osseous elements of the nose to give support to the bridge is the important factor for our consideration, and our plan is to produce an elongated nasal bone with ossification to the nasal process of the frontal bone. This can be accomplished only by elevating the periosteum and transplanting the upper end of the graft, which will be taken from the tibia, beneath the periosteum of the frontal bone and the periosteum that remains on the remnant of the nasal bone.

That is the plan for the preparation of the bed for the graft, and now we will follow out this scheme by incising the tissue in the midsubciliary ridge down to and through the periosteum. This incision is made parallel to the long axis of the nose, and is about ¾ inch in length. The periosteum is now elevated with a raspirator down to the tip of the stump of the nasal bone. A scissors curved on the flat is now used to split the nasal structures on the flat, endeavoring to keep midway between the mucous and subcutaneous surfaces, so as not to open either below the primary incision. A slow subcutaneous cutting dissection is made with the tip of the scissors clear down to the tip of the nose. The space is about ¾ inch in width all the way. It is a little broader at the tip than higher up. Now the bed for the graft is prepared, and we have been fortunate enough not to have opened the mucosa of the skin so that our graft can be completely covered, otherwise we could not have proceeded further with the operation.

You will note that we have made the incision high on the forehead, so that the lower angle would not be low enough to be subjected to lateral traction over the graft as it gives support to the skin.

We will now prepare the graft from the tibia. Never make the incision directly over the spine of the tibia, but place it to the outer side, over the tibialis anticus muscle. [Removes graft from tibia and adjusts it to the nose.] From now on the result depends on the asepsis of our work. The nasal and frontal bones having been freshened to receive the graft—it goes into its new bed without difficulty. The periosteum is now sutured over the graft, so that its upper end is practically surrounded by osteogenetic influences. The skin is closed by horsehair sutures.

You will note that I was very careful to contact the graft with the living bone of the nose, otherwise the graft would be absorbed. That was one of the lessons we learned very early in connection with bone-grafting. We had one case, in 1876, in which a thumb was transplanted onto a nose—the one patient had a double thumb and the other a saddle-nose. We took the supernumerary thumb, transplanted it onto the nose, and had a beautiful operative result, but it was all for naught. At the end of fourteen months it commenced to get smaller, and from then on it diminished rapidly in size until there was nothing left, just because I did not contact the bone of the thumb with the living bone of the face. In addition, however, it was a homograft. Paraffin has been used with fairly good cosmetic results, but occasionally the skin becomes thinner and thinner over the paraffin, until it is exposed and must be removed.

If the graft survives in this case, as it should, owing to the slight pressure, and does not absorb, the result should be excellent.

Let the record show that the hemorrhage has been controlled; that we made a division of the tissues, then tunneled under the periosteum to the frontal bone, making an incision parallel to the long axis of the nose; that we then extended this denudation down to and including the nasal bones as far as the tip, this being done with the sharp gynesic scissors (Fig. 163, B); that a bone-graft of proper size and shape to make the elevation was taken from the tibia (Fig. 163, C) and passed in; that it was cut at a sharp angle in its upper portion, so as to fit into the groove on the frontal bone; that it spanned the depression of the nasal bones (Fig. 163, D); that the periosteum was elevated on the nasal bones laterally; that in inserting the graft the periosteum faced upward; that the graft, with its sharp ledge, was inserted so that it rested against the frontal bone (Fig. 163, D), from which we expect osteogenesis, and that the graft measured 4.5 cm. in length.

That is a good nose if the graft survives. You see the strong leverage above and below will keep the graft in contact with the

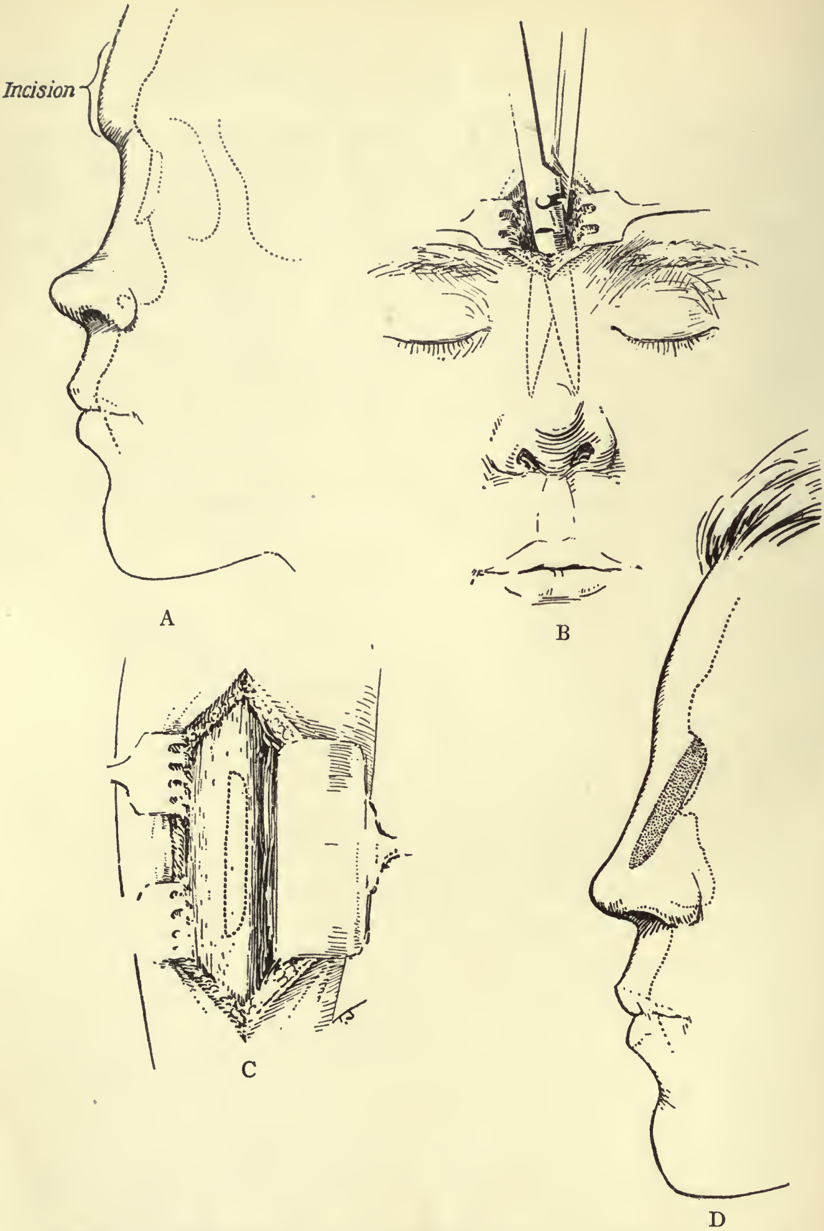

Fig. 163.—Plastic on nose: A, Profile view of saddle-nose deformity; relation of skeleton to soft parts of face inserted in dotted outline; incision over glabella shown; B, edges of incision retracted; fine scissors inserted through incision dissect subcutaneously as far as tip of nose: they avoid opening mucosa; C, subcutaneous surface of tibia exposed preparatory to removal of bone-graft, whose pattern is shown in dotted outline; after removal the graft is pared to its proper shape and inserted into wound subperiosteally; D, profile view after reconstruction of skeleton of nose; shows bone-graft in situ. Its upper end contacts with the frontal bone. The upper part of its posterior surface contacts with the remnants of the nasal bones. The lower portion of the bullet-shaped graft rests upon the cartilaginous skeleton. The lower end of the graft supports the tip of the nose.

nasal bones, so that osteogenesis from them will be conducted right into the graft.

[NOTE.—Primary union took place. The graft healed in nicely, and on June 25th I received a report from his physician saying that there had been no untoward symptoms and that the nose looked normal.—ED.]

CARCINOMA OF LOWER LIP AND OF SUBMAXILLARY LYMPH-NODES—EXCISION OF CANCER-BEARING AREA—PLASTIC RESTORATION OF LIP

Summary: Clinical history of patient; examination by Dr. Murphy; comments—cancer of lip, so visible, neglected for hidden cancer elsewhere in body; statistics upon prognosis; procrastination in appendicitis not tolerated by laity; passage of control of sanitary matters from profession to laity; the greatest cause of mortality in surgery to-day is procrastination; recent statistics indicate necessity of wider excision of tissues; Handley's demonstration of the mode of dissemination of cancer-cells; requirements of a successful operation; three sets of lymph-nodes that may become involved, and prognosis based upon their involvement; operation.

HISTORY

The patient, a male, married, aged forty-nine years, was admitted to the hospital September 2, 1914. The family and past histories are negative.

The present trouble began eighteen months ago, in March, 1913, as an abrasion of the skin of the lower lip. Four weeks later three or four small warts had sprung up at the same spot. He consulted a physician, who told him that this condition would eventually disappear. Six weeks ago, in March, 1914, he consulted another physician who cauterized the warts with an acid which caused a swelling. By this time the warts had coalesced and formed a lump with a fissure from which there was yellowish discharge. He was given an ointment to apply, and also hydrogen dioxid to cleanse the wound. The tumor is now the size of a pigeon's egg (Fig. 164). The left submaxillary lymph-node is enlarged.

Examination by Dr. Murphy on August 31, 1914: Let the record show that the submaxillary lymph-node on the left side is adherent and fixed to the bone: that is, the node which is located in the anterior pole of the submaxillary salivary gland; that there is no evidence of thickening over the foramen menti; and that an extensive dissection must be performed, removing all the lip and part of the chin, as well as the involved lymph-nodes.

Dr. Murphy: This is another example of the results of expectant treatment. In perusing the literature for the last six or eight years one finds that we have been removing carcinoma of the esophagus; that we are discussing and giving great attention to the removal of carcinoma of the cardiac end of the stomach; that we are endeavoring to take out tumors of the hypophysis, and yet are allowing epitheliomata of the lip to become fatal every day! That is the irony of the practice of medicine. Every man knows that is true. It is sad. It is lamentable that it should be true, because carcinoma of the lower lip is readily recognized, and the differential diagnosis from syphilis, tuberculosis, and other likely lesions is easily established. We neglect them until the condition develops in which this man appears to-day, with a tumor the size of a pigeon's egg (Figs. 164 and 165). In the report of St. Bartholomew's Hospital in London, which contains an analysis of the cases for twenty-five years, we note that the cases operated upon *before* demonstrable enlargement of the superficial lymph-nodes showed a final cancer mortality of 52 per cent. Of the cases operated upon in which the nodes were palpable, and in which the nodes had been removed, 76 per cent. died a cancer death, which proves that it is an extremely malignant disease. While the disease is right out in the open, exposed to the observation of the physician and the patient's friends from the first day, still we rarely receive the cases early for operation. The appendicitis proposition has been settled by the people, as illustrated in that patient last night whose friend would not tolerate procrastination. Why? Because he had been educated by the public press that that was the wrong thing to do. The hope of cancer rests in the education afforded by the public press. It is just the same in sanitary matters, which, fortunately for the people, have practically passed out of the control of the medical profession, and now are carried out effectively by the laity. Laymen are filling these sanitary positions, and yet it should not be so. We know better as a body and we know better as in-

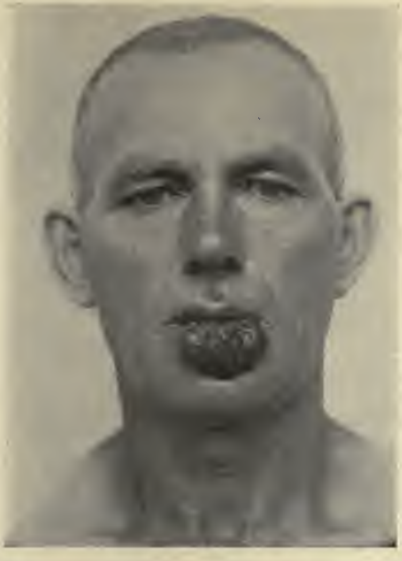

Fig. 164.—Carcinoma of lower lip (front view). (See p. 600.)

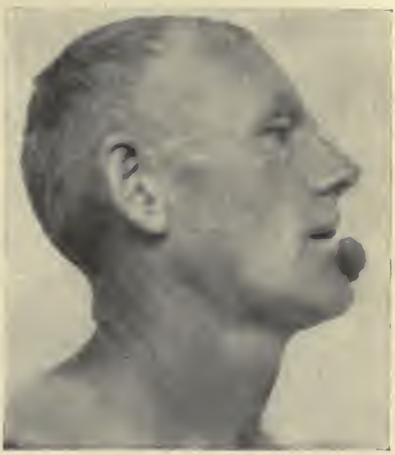

Fig. 165.—Carcinoma of lower lip (lateral view).

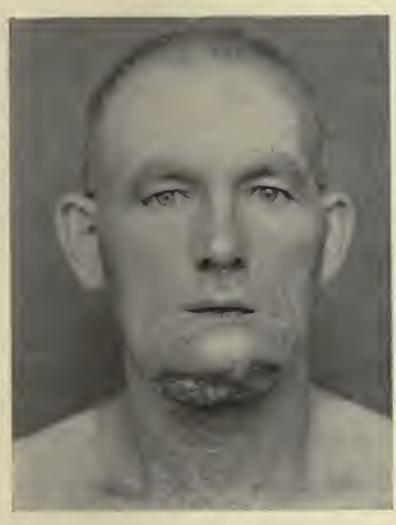

Fig. 166.—Carcinoma of lower lip after operation (front view). (See p. 601.)

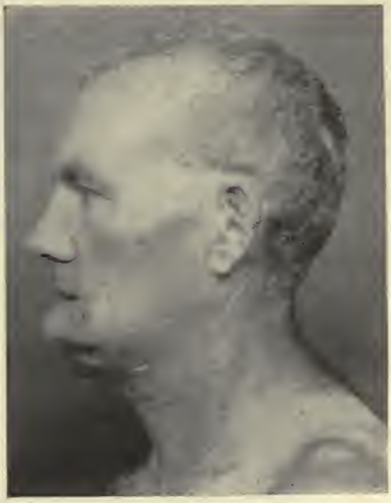

Fig. 167.—Carcinoma of lower lip after operation (lateral view).

dividuals, but still we procrastinate! Some one asked us not long ago what was the greatest cause of mortality in surgical lesions. We said: "The greatest cause of mortality in surgery to-day is procrastination. It causes a greater mortality than cancer." Individually, the people dislike gross surgical procedures as well as radical cures.

VISITING DOCTOR: What is the result of this procrastination on the part of the medical profession?

DR. MURPHY: Dissemination of disease to wider areas.

From the more recent statistics on the transmission of carcinoma we have concluded that a larger area of skin and of tissue in the neighborhood of the lesion must be removed in the primary operation. We believe that most of the dissemination may be due to the leukocytes' or lymphocytes' picking up the cancer-cells and depositing them in the radicles of the regional lymphatics. We think Handley has proved rather conclusively that the malignant epithelial cells are pushed into the lymph-spaces by the growth of the cells beneath piling the one above the other. At the last Clinical Congress of Surgeons of North America he demonstrated this, and also how the cancer-cells are then carried through the lymph-vessels and arrested in the next lymph-node.

When we come to apply these principles to this case of carcinoma of the lip, it means that we must excise a wide area; that penetration in the lower lip must be downward, because the orifice of the mouth bounds the lip above; and that there is a cancerous filling of the lymph-spaces in addition to the primary carcinoma, with an arrest of the cancer-cells in the lymph-nodes.

Three sets of lymph-nodes may become involved. First, and the most commonly overlooked, is the lymph-node at the foramen menti; second, the node situated in the anterior pole of the submaxillary salivary gland; third, the vertical chain of lymph-nodes, which extends downward. This patient already has involvement of the submaxillary lymph-node, which is fixed to the mandible—always a serious condition.

What is the prognosis in this case? The chances are 90 per cent. that he dies a cancer death, and we give him the advantage of every possible ray of hope. Look at the size of that lesion standing up like a big red flag! Look at the size of that node! That is not a "country doctor's" patient: it is a "city doctor's" patient.

[During the course of the operation Dr. Murphy emphasized the following points:

1. Uses hemostats with weak spring so as not to crush the tissues.

2. There is no necessity of removing the whole submaxillary salivary gland, but only that portion (the anterior pole) that contains the involved submaxillary lymph-node.

3. Demonstrates deposits of cancer at foramen menti.

4. Saves all mucosa of lip possible.

5. Fashions new lip from skin over chin.

6. Does not interfere with circulation of flap. Have regard for vessels and do not subject flap to too much tension.

7. Key to good work in plastic surgery of lip is restoration of mucosa with a perfectly movable lip.]

Let the record show that we made a square-cut division transversely; that we made an incision underneath the submaxillary salivary gland, loosening the skin and bringing it up across the midline so as to give us a full mucous lining for the flap; that we removed the submaxillary salivary gland, and that we brought up the skin and tissue of the chin to construct an upper lip. Let the record show further that the submaxillary lymph-node was not adherent to the periosteum, as we had concluded from physical examination (Figs. 166 and 167).

The microscope revealed a basal-cell carcinoma.

[A case of carcinoma of lip with extensive comments was published in the Clinics for August, 1912, pp. 571–580; one involving the upper lip, and starting in an old lupus scar, with sketches of steps of operation, was published in the Clinics for April, 1915, pp. 289–294.]

EARLY CARCINOMA OF LOWER LIP—RADICAL EXCISION

Summary: Clinical history of case; comments—danger of a scar at an orifice; trauma-relationship of cancer and of sarcoma; early stage of this case on admission exceptional; time of metastases to other organs; steps of operation; a swelling is nature's method of coffer-damming; danger of injury to an exposed branch of facial nerve; difference in clinical course between malignancy and infections, and difference in pattern of cellular metastases; in neoplasms cells make mischief: in infections, bacteria; description of submaxillary lymph-node; postoperative discourse—time malignant cell passes from one node to another; squamous-cell cancer and not adenocarcinoma attacks lip.

HISTORY

The patient, a male, single, aged thirty-nine years, was admitted to the hospital September 14, 1914.

The family and venereal histories are negative. He had the usual diseases of childhood, and, fourteen years ago, an attack of typhoid fever of three weeks' duration. Ten years ago, in 1904, he was kicked in the face by a horse, injuring the nose *and both lips.*

The present trouble began one year ago, in 1913, with scab-formation on the lower lip, between its angle and the median line, and at the mucocutaneous junction. He scratched it repeatedly, removing small crusts. The under crust felt indurated. After applying an ointment a few times it seemed better.

In the last six months there has been no change. A scab forms and is scratched away. The induration is about the size of a small bean. Aside from the formation of a small scab, the lesion has never annoyed him. There are no palpably enlarged lymph-nodes in the neck.

COMMENTS AND OPERATION

Dr. Murphy: Wherever you have a scar in any of the orifices, it is a source of danger. This patient cut his lower lip, and the scar is a source of irritation. Carcinoma is the sequence of repeated mild irritations—this is a beautiful case of repeated mild irritations. That etiology differs materially from sarcoma. Sarcoma is the sequence of a moderately severe trauma, never of a severe trauma. In other words, you never get a sarcoma at the site of a fracture. You may have a sarcoma and then a fracture, but you never get a sarcoma where the bone is broken. But where you hit the bone with a baseball, with a bat, or strike it against the edge of a chair, stopping short of fracture, that is the type of fracture that produces sarcoma. Do you grasp the difference in the etiology of these two neoplasms? Take carcinoma of the stomach: it is the result of repeated irritations, and the stomach is the most common site of carcinoma since we have learned to diagnose the latter. This patient's lesion has never disappeared since it was first noticed. It improved, got worse, but never got well.

This laceration of the lower lip was made by a horse kick, just at the mucocutaneous junction, and in shaving close to it the edge of the razor frequently cut it. Some hair grew in the wrong direction, and in shaving he occasionally took off a bit of tissue from the lip and it bled. See how accurately the etiologic factor is brought out by the patient's story. You remember the other case started from a lupus, and in shaving the patient took off a little scale with the razor. [CLINICS, April, 1915, p. 289.] Notice the repeated mild irritations. The same sequence occurs in the tongue—repeated tooth irritation and finally malignant growth. By recognizing the significance of these mild irritations we should be able to do good prophylactic work.

This patient comes to us in the superlatively early stage of the disease. That is just the opposite to what happened in the preceding case. *He was not sent in by a doctor—he came in!* He quoted the physician as saying that the trouble did not amount to anything, and just to keep on applying the salves! This is, unfortunately and lamentably, the management of this class of cases.

[Dr. Murphy here refers to the time of metastasis to other organs, citing the case of the Japanese waltzing mouse. This is given on p. 701.]

[Dr. Murphy excises the mucosa bearing the cancer.]

In his estimate of cancer, Handley believed that the cells spread equally in all directions through the same tissue—that they do not cross the line into a different tissue, as a muscle. Therefore, we excise the loose mucosa, since the cells do not pass across the tract into a different type of tissue. A physician can perform this operation in his office when he does it early, but when he waits until he has the type of operation you saw me perform upon the previous case, then he has a major operation in place of a minor. Now, in the reconstruction of this lip, we first sew the mucosa accurately on the inner side with an entropion of its edge, not including the muscular layer. Our object is to reconstruct the lip to the same thickness as before. Now, the next step is to approximate the muscular layer so that, instead of showing a depression at the scar line, the lip will present an elevation, which will level itself when absorption takes place. [Dr. Murphy cites the cancer statistics of St. Bartholomew's Hospital. Consult preceding case, p. 600.]

This was a minimum manifestation on the lip, scarcely recognizable as a carcinoma, with a maximum manifestation in the lymph-node. The fact that the lymph-node has reacted to the irritation is in favor of its retaining the microörganisms or the parasitic cells, as the case may be. We used to consider a swelling around an infected focus of any kind detrimental: we poulticed and treated it—in fact, we did everything to banish the swelling. But now we know that the swelling is an announcement of the efficiency of nature in coffer-damming the infective microörganisms and their products. It is the effort on the part of the tissues to imprison the harmful agent and prevent its spread. [The supramandibular branch of the facial nerve is liable to be cut, owing to its low drop, resulting not so much in a dropping as in an expressionless lip.]

Let us analyze the difference in the clinical course between malignancy and infections, seeing how far they are alike and how far they diverge. Take, for example, tuberculosis. First, this disease is a local manifestation. It destroys by the multiplication of cells, as in carcinoma: there is a local center of necrosis, just as in carcinoma. Second, it is transmitted to the neighboring lymphatics. Third, it may be transmitted to the bloodstream and to other positions in the body. Fourth, it destroys life.

These similarities between malignant disease and infections are not continued in the pattern of the cellular metastases. In malignant disease the metastasized cells always reproduce the pattern of those at the primary focus. For example, if you have a squamous epithelioma of the lip and a metastatic cancer appears in the femur, it would be a squamous epithelial cancer. In infection, on the other hand, it is the cells of the tissue in which the infection lodges that are active. If you have a tuberculosis of the lip and get a tuberculosis of the femur, there would not be epithelial cells, but osseous cells, at the point of infection. It seems to me that this is a proposition of cells. The cell is the destructive element both in sarcoma and in carcinoma; the microörganisms are the destroyers in all other varieties of infection. The biotic and toxic effects of the microörganisms cause the changes that occur with infection. The multiplication of cells produces the change that occurs with malignant disease.

Let the record show that the submaxillary lymph-node was enlarged to four times its normal size, and was three times its normal transverse diameter and twice its normal thickness; that it was red, congested, and infiltrated; that it was enucleated from its position in the apex of the submaxillary salivary gland, and that it was not adherent to the bone (Fig. 168).

Here is another factor which we do not know except empirically. How soon after the malignant cell is carried into the first lymph-node does it invade the next? We have not solved this problem experimentally or in any other way. Remember, in a lymph-node there are two terminal circulations, one afferent and the other efferent. For example, in carcinoma of the pylorus a metastasizing cell passes into either the superior or the inferior gastric lymph-nodes and later may be found in the supraclavicular lymph-node. But before the malignant cell arrives

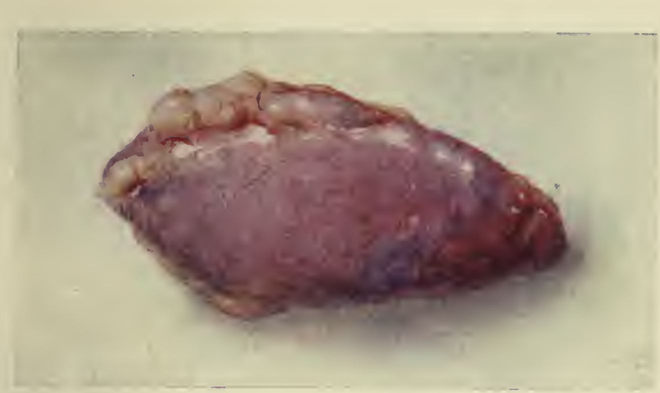

Fig. 168.—Secondary carcinoma of the submaxillary lymph-node situated at anterior pole of submaxillary salivary gland. It is four times the normal size, notwithstanding the small size of the labial epithelioma. Despite its size, it was not even palpable. This node forms the first filter for the lymphatics from the lower lip.

there, it has to pass through three, four, or five different lymph-node filters in the mediastinum, so that not infrequently in carcinoma one finds nodes involved at intervals along this route.

[The pathologist just reports to me that the node removed shows an adenocarcinoma.] In this we differ. It should not be an adenocarcinoma. It should be a squamous-cell carcinoma. If that is an adenocarcinoma, it is the first I have seen in the lip in my entire practice. I think he is in error, because an adenocarcinoma in that situation is rare. I shall show you how to check up. If it is an adenocarcinoma, the cells in the infected nodes will not be squamous in type. They will be adenocarcinoma cells. If it is a squamous-cell carcinoma, the secondary neoplasm will be squamous celled. *He* will check it up again. That lymph-node is almost completely surrounded by tissue. The pathologist did not know the story, but his interpretation of the cells is erroneous. *They must be squamous cells!*

[September 21, 1914: The pathologist's (Dr. Sweek's) final report on specimens fixed and run through by the paraffin method is that the tumor is a basal-cell carcinoma.]

OSTEOMYELITIS OF THE MAXILLARY ANTRUM

Summary: Clinical history of the patient; operation discloses cause of chronicity of discharge; thorough drainage established by enlarging maxillary ostium; hemorrhage controlled by gauze packing; comparison of maxillary antrum disease with that of the other sinuses; demonstration on a skull of the steps of the operation.

HISTORY

The patient, a female aged thirty-eight years, married, came to the office April 6, 1915, presenting a swelling of the soft tissues over the right maxillary antrum, and a constant discharge of pus through the opening made into the antrum at a previous operation. Her family, past, and menstrual histories are negative.

In August, 1909, nearly six years previously, she had a number of teeth capped. There was no history of infection at the time. One year later, in August, 1910, she noticed a small enlargement in the region of the right antrum, which for over three years slowly increased in size until by April 1, 1914, it was the size of an almond. Operation on this date revealed a bone cyst, and the antrum was opened. The face was markedly swollen, but within a month it subsided to its present size, which it has not exceeded since. On April 5, 1914, four days after the operation, it began to discharge pus through the mouth, and this discharge has continued without interruption. Six months later, on October 6, 1914, on account of the continued swelling and pus discharge it was cureted again without effect. Ten weeks after this, on December 18, 1914, there began sharp pain in the right half of the face. There was no fever at this time. One week later, on December 24th, escape of pus from the nose relieved the pain. Since then there has been a discharge into both the nose and the mouth.

In March, 1915, an abscess formed at the point of swelling, incision of which brought forth a sequestrum. She has been having irrigations through the nose, but despite these the swelling has remained, discharging pus into the mouth and nose.

A skiagram revealed trouble at the root of a tooth, for which she had been referred to a dentist before anything could be done in a surgical way. She returned on April 22, 1915, the dentist having filled the root and taken a skiagram to see that it was all right. She was advised then to go to hospital and have the antrum opened. Examination of the mass on the right side shows that it is soft and fluctuating.

COMMENTS AND OPERATION

Dr. Murphy (April 27, 1915): Let the record show that there was a lot of carious bone here; that the posterior cavity was shut off entirely; and that the whole of the inner lining was carious, and the opening into the anterior nares closed.

Let the record also show that a carious piece of bone protruded at the upper angle of the cavity, obstructing the ostium; that this is what kept up the constant discharge; that we drilled an opening through the middle meatus, which was filled with granulation tissue, enlarging it enough to admit the tip of the index-finger right into the nose, and then inserted a strand of nosophen gauze through the antrum and the enlarged maxillary ostium into the nasal chamber, so as to control the hemorrhage and prevent its entry into the pharynx. We shall let that packing remain *in situ* until the bleeding stops. The procedure was not difficult.

Of the various sinus involvements, those of the maxillary antrum are the worst, with the exception of the frontal sinuses. I never have an opportunity to do this work primarily: it is only when some complication occurs that it comes to me. Now I shall demonstrate on the skull just what we did. Here you see the maxillary antrum. Now then, in going through, you see how we come to the left. The osteoma had developed up here, separating this cavity from the posterior cavity, and keeping it suppurating. We burred away this piece of bone in front until the whole finger went through into the nasal chamber. I think it will get entirely well, and do not believe the osteoma will recur.

May 20, 1915: There is but little pus discharging now. The patient is feeling much better.

TRAUMATIC EPILEPSY.—REMOVAL OF HEMORRHAGIC EPIDURAL CYST

Summary: Clinical history of patient; explanation of low blood-pressure—of rapidity of pulse; analysis of symptoms; difficulty of diagnosis of skull injuries; differential diagnosis; operation—importance of careful hemostasis; minute craniocerebral topography unnecessary in these days of osteoplastic flaps; instruments for cutting through cranium; demonstration on table of middle meningeal hemorrhage; epidural *vs.* subdural hemorrhage; hemorrhage by *contrecoup;* method of ligating middle meningeal artery; case citation showing fatal rapidity of hemorrhage; immediate control by ligating external carotid; exposure of cysts; contents of hemorrhagic cysts; correlation of clinical with operative findings; occurrence of fat embolism; management of fracture of humerus.

HISTORY

The patient is a married woman, aged fifty-two years. Her family history is negative as to carcinoma. One son died of pulmonary tuberculosis at the age of twenty-four years. He was sick for only two months. Her menstrual and past histories have no bearing on her present trouble.

On August 28, 1914, while crossing a railroad track in an automobile, the machine was struck by a locomotive, just as it had nearly cleared the track. The patient was sitting in the rear seat, directly in the path of the locomotive. She was thrown out of the automobile and at some distance from it, but does not know exactly how far nor in what way she landed. The patient thinks she must have been struck on the head by the locomotive, since she has no remembrance of the impending accident nor of being thrown. On her arrival at Mercy Hospital (not on Dr. Murphy's service) two hours after the accident she was delirious and talkative, but oblivious to her surroundings.

On examination it was found that she had sustained a compound fracture of the left humerus just above the elbow-joint. The humerus was protruding through the skin for a distance of about two inches (Fig. 169). She could move the left arm at the shoulder, and could also move the fingers of the left hand. The radial pulse in each wrist was equal and synchronous. There were three scalp wounds. One was located in the left temporal region, back of the ear, and extended clear down to the bone. There was another, two inches long, over the occiput, and a third, about one inch long, on the back of the head, somewhat to the right of the median line. The pupils were equal and reacted promptly to light. There was no hemorrhage from the ear, nose, or mouth, and no ecchymosis visible either in the orbit or elsewhere. Nevertheless, a presumptive diagnosis of basal skull fracture was made.

The wound of the left arm (compound fracture) was swabbed out with iodin and the elbow was supported upon an angular splint. The scalp wounds, too, were swabbed out with tincture of iodin, and a dry gauze dressing was applied. Adhesive plaster was used to bring together the edges of the scalp wound.

The patient was delirious for three days after the accident, moaning considerably, apparently from pain. She was catheterized the day after the accident. The urine contained no blood. The bowels moved normally, and the stools contained no blood. On the day after the accident the patient had at times slight twitching of the muscles of the face and of the right arm. At 10 A. M. on September 7, 1914, ten days after the accident, the patient had an attack resembling *petit mal.* She experienced a sense of pressure on top of the head and lost consciousness for a few seconds. There was a marked twitching of the right arm and of the muscles of the face, especially on the right side.

Since the initial attack similar ones have occurred several times, both by night and by day. Their duration varies from five to twenty minutes; and the interval between them varies from twenty minutes to forty-eight hours. The patient always has a subjective prodrome of some sort—a sense of weakness, a sense of pressure over the head, or pain on the left side of the head.

Examination of the eyes by Dr. Tivnen, on September 20, 1914, showed no evidence of choked disk in either eye. The patient is slightly deaf in the left ear to the watch test. She thinks she heard well before the accident. She has no nystagmus and she has not complained of attacks of dizziness at any time.

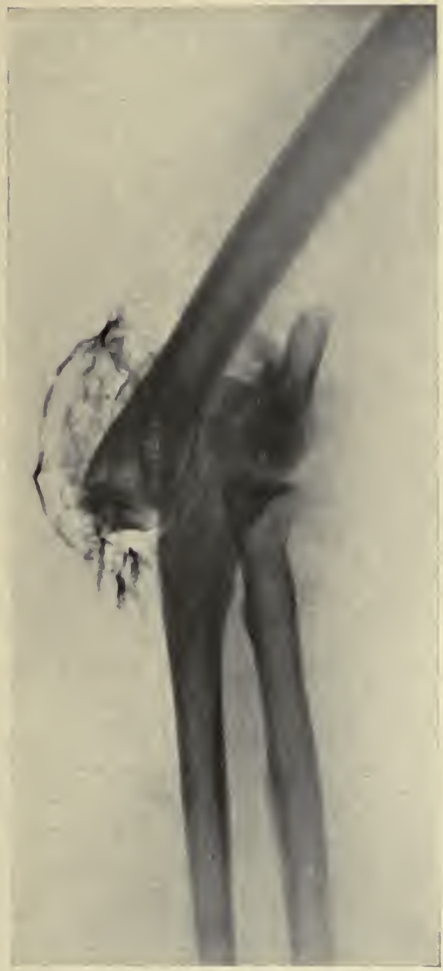

Fig. 169.—Compound fracture of the lower end of the left humerus involving the joint. Note the extensive shadow thrown by the newly formed osseous tissue about the joint. Note that Dr. Murphy's conservative treatment has thus far prevented the development of any osteomyelitis in this compound fracture (See p. 615).

COMMENTS AND OPERATION

Dr. Murphy (September 28, 1914): The patient comes under my treatment one month after the accident. Dr. Mix has gone over this patient, and I shall ask him to give you the results of his examination.

Dr. Mix: The patient was struck on the head at the time that the automobile was hit by the locomotive. She suffered three very severe scalp wounds, which are described in the intern's history, just read. The largest of these wounds, the one which is doubtless responsible for her present symptoms, was over the lower left side of the head. Its location is indicated by the present scar.

She was admitted to the hospital about 12.15 on the morning of August 28, 1914. At the time of her admission she was in deep shock and was vomiting. The vomiting continued during the rest of the night, and the vomitus consisted of mucus and bile. She was partially unconscious, according to the nurse, during almost all the night; at least, she was in a condition of marked stupor, although it was possible to arouse her somewhat. Her systolic blood-pressure was taken and was found to be only 115 mm. There was no dilatation of the pupils. In other words, the usual signs of epidural hemorrhage, namely, dilatation of the pupils and high blood-pressure due to increased intracranial pressure, were missing, although vomiting was present.

With an increase in intracranial pressure one expects a corresponding increase in blood-pressure unless there be some concomitant reason for the reduction of the latter. A high blood-pressure, in fact, is commonly caused by only three pathologic conditions. It is well to know what these conditions are. The first is nephritis, particularly the contracted kidney of chronic nephritis. The second condition is arteriosclerosis, located chiefly in the large arteries of the trunk, especially in the mesenteric and renal arteries, for arteriosclerosis of the small arteries of the extremities alone does not raise the blood-pressure appreciably. The third condition is increased intracranial pressure from any source whatever, whether an accumulation of blood, an accumulation of cerebrospinal fluid, or a neoplasm.

One would have expected this woman to have had increased intracranial pressure on entering the hospital, provided that at the time of the accident she had acquired a subdural hemorrhage or a laceration of the middle meningeal artery, or of one of its branches, with an epidural hemorrhage. Yet her blood-pressure was only 115 mm. on admission to hospital. Must we, therefore, because of this low blood-pressure, rule out the possibility of an intracranial hemorrhage in her case?

It is perhaps possible that there may have been enough shock caused by the accident and enough hemorrhage from the compound fracture of the elbow to counteract or to neutralize for a time the tendency toward a high blood-pressure, even in the presence of an accompanying intracranial hemorrhage. The effect on the symptoms of a compound fracture of the left humerus occurring simultaneously with an injury to the brain is something difficult to calculate on *à priori* grounds. A cerebral injury, provided there was an escape of blood in some amount, would tend to increase the blood-pressure and diminish the pulse-rate. A fracture of the left humerus would tend to diminish the blood-pressure and to increase the pulse-rate. It is, perhaps, possible that the contrary effects of these two lesions may have neutralized each other, so that the present symptomatology departs from the usual and customary.

The pulse-rate has not been such as one would expect to find with increased intracranial pressure from hemorrhage. Increased intracranial pressure slows the pulse and causes a bradycardia; but this patient's pulse has increased in rate. On admission, the rate was 92; at 2 A. M., 92; at 4 A. M., 96; at 6 A. M., 100; at 8 A. M., 110; at 10 A. M., 120; at 12 M., 120; at 2 P. M. on August 28 it was 120. Instead of showing the usual signs of increased intracranial pressure from hemorrhage, namely, increase of the blood-pressure, slowing of the pulse-rate, and dilated pupils, she had none of these signs. She had only the vomiting, which is associated frequently with shock also, and which, therefore, could be construed as being due either to a lesion of the brain itself or to "surgical shock."

Early the morning after the accident, about 8 o'clock, that is, about eight hours after the patient's admission to hospital, and not more than ten hours after the accident, the nurse recorded an exceptionally important observation, that is, a *twitching* of the muscles of the right side of the face and of the right arm. I talked to the nurse about this record myself, and she was positive that the twitching was confined to the right side of the face and the right arm. Such localized convulsive movements indicate an irritative lesion involving the left precentral convolution, where the centers for the right side of the face and for the right arm are located. This cortical area corresponds with the position of the scalp wound which the patient had on the left side of the head.

Such localized convulsive movements, when not associated with loss of consciousness, constitute Jacksonian epilepsy. In this case the epilepsy was not truly Jacksonian in the sense that the patient, in a state of complete consciousness, could contemplate the seizure and describe it accurately to me subsequently; but it was Jacksonian in the sense that it was a localized convulsion, a characteristic feature of focal lesions.

It is probable that the clouding of consciousness was gradually increasing at eight o'clock in the morning, about the time that this Jacksonian attack appeared, for the patient subsequently passed into a condition of marked stupor, according to the nurse's report. These two manifestations, namely, the presence of the Jacksonian seizure, together with the coma deepening some hours after the original injury, are the most important facts which the history of this case brings to our consideration. The Jacksonian attack gives us as the localization of the brain lesion the cortical center for the right arm and the right side of the face, that is, the left precentral gyrus. The increasing loss of consciousness suggests the etiology, namely, a subdural hemorrhage due to gradual oozing from one or more branches of the middle meningeal artery.

When the middle meningeal artery is lacerated by an injury, consciousness is not immediately lost. It is usually gradually interfered with. There is a varying period of time during which consciousness persists until the accumulation of the blood poured from the artery is extensive enough to cause sufficient pressure upon the brain to destroy consciousness. In middle meningeal hemorrhage due to trauma we find, as a rule, that consciousness is at first present, but that after a shorter or longer period of time, depending upon the rapidity of hemorrhage and of compression of the brain, loss of consciousness supervenes. This patient lost consciousness during the morning of August 28. She was moaning when brought to hospital at 12.15 A. M.; but by the following morning she was in a condition of mild stupor.

She remained in this state, according to the nurse, for a period of about two days, vomiting constantly during the day after the accident. It is stated on the nurse's record sheet that the patient vomited five ounces of fluid at 10 A. M., three ounces at 11 A. M., at 3 P. M., and again at 8.30 P. M. The note is made that she was apparently dozing at noontime, and seemed to be sleeping at 8 P. M. I find, however, on questioning the nurse, that this was not a normal sleep, but rather a mild degree of stupor.

There was another important symptom noted which is frequently present in cerebral injuries, namely, suppression of urine. The patient passed no urine on the day after the injury, and "had to be catheterized" on the morning of the twenty-ninth. At this time 920 c.c. were removed. Again, the same evening, at 6 P. M., she was catheterized, and 450 c.c. of urine were removed.

On August 29 the patient's pulse-rate began to fall. That the previous rapid pulse-rate was probably due to shock is indicated by the fact that, as the shock passed away, the pulse-rate fell to 100 at 3 A. M. on the twenty-ninth; 90 at noon; 94 at 3.35 P. M.; 88 at 6 P. M., and 84 at 9 P. M. This fall in the pulse-rate, to my mind, is best explained as due to a gradual subsidence of the condition of shock rather than to an increase of intracranial pressure from continued intracranial hemorrhage.

There is a note that the patient spat up a little bloody mucus at noon on August 29, thirty-six hours after the accident. Since

I did not see the patient at the time, I am unable to state the source of this bloody sputum, and the nurse is unable to give us any definite information on this point. Whether the blood came from the pharynx or from the lungs we do not know; and, so far as I am aware, this is the only time that she spat up any blood. Examination of the chest showed no abnormality.

On August 30 the sphincter of the bladder began again to functionate normally. The bowels, which had moved only on the first day after the accident by using an enema, began moving normally August 31, two days later, apparently of their own accord. On this third day of the illness the pulse-rate remained low, but did not, however, for some days pass below 80.

Since the original attack described in the intern's history, several spasmodic seizures confined to the right arm and the right side of the face have been noticed. Dr. Kelly, who has observed the patient very closely in these attacks, made an observation which to me was unique. He found an area on the left side of the scalp, by pressing upon which he could change the location of the convulsive twitchings from the right arm to the right side of the face. On relaxing the pressure the face ceased twitching and the arm began to jerk again. During one convulsion he brought about this change six times, the patient being totally unconscious all the time. This observation, repeatedly verified, points very definitely to an intracranial lesion in the immediate vicinity of the injury to the scalp.

I feel justified in concluding that at the time of the automobile accident there was a laceration of some of the smaller branches of the middle meningeal artery on the left side of the skull, and that an epidural or subdural hemorrhage occurred over the centers for the arm and face, thus affecting the motor functions of the right half of the body. I believe the reason that the patient did not immediately become completely unconscious was that there was at first not a sufficient accumulation of blood to produce complete coma from pressure; but as the blood continued to ooze out the intracranial pressure gradually increased until the patient finally passed into a profound stupor.

The diagnosis in this case is still further complicated by the husband's statement that twenty years ago the patient, while giving birth to a child, had a convulsion. We do not know whether or not that convulsion was eclamptic. Probably it was not, because, during her girlhood and before marriage, she had been a sufferer from convulsions. The question which confronts us at the present time is whether these old convulsions have anything to do with the present Jacksonian epilepsy. My opinion is that the two conditions are distinct, and that the injury which she suffered recently led to a subdural or epidural hemorrhage, with the corresponding localized cortical pressure disturbances.

When the skull is opened now, I do not believe that any large clot will be found. Sufficient time has already elapsed for the absorption of any clot which previously had formed. There has been time enough for the dura to become much thickened by organization of the clot. Probably it will be found to be much thicker than normal.

After these hemorrhages the hemorrhagic areas usually clear up fairly well, and unless an operation is done very soon after the injury, the only thing found subsequently is a thickening, sometimes of very considerable degree, even to more than twice the ordinary thickness of the dura. Although a serous or a hemorrhagic cyst *may* form following such a hematoma, our experience is that it seldom does.

The scalp injuries, now that the head has been shaved, show perfectly clearly. This injury here, I believe, caused the present syndrome [indicating the scar on the left side of the head], particularly the Jacksonian seizures.

Dr. Murphy: Dr. Mix has mentioned the fact that the patient had some convulsions previously. This is one of a number of factors which complicates the diagnosis of the case. Diagnosis, I believe, is the most important and the most difficult part of the surgery of skull injuries. You can see how relatively simple our task would be in this case if we were only perfectly certain in advance of exactly what is the matter with the patient. This statement holds not only for injuries of the skull, but just as well, or even better, for injuries of the abdomen. In general, the most difficult task in emergency surgery, I believe, is the making of correct and early diagnoses. It is a great help to us to have Dr. Mix's clear-cut analysis of the case.

Dr. Mix has stated that the possibility of a fracture of the base of the skull was considered on the patient's admission to hospital, but was ruled out clinically because of the absence of the essential symptoms of a fracture of the base. The essential symptoms of a basal fracture are bleeding from the ear, mouth, or nose, ecchymosis behind the ear (Battle's sign), beneath the conjunctiva, and in the orbital region. Lastly, and very suggestive in the diagnosis, is a marked lowering of the pulse-rate. The pulse may slow down to 50, 40, or even as low as 32 beats a minute. I remember the case of a fireman who fell from a ladder quite a distance and struck on his head, producing a basal skull fracture. For three weeks his pulse-rate was as low as 32.

Here is the scar beneath which we expect to find the pathology [indicating]. It is possible that a previous epilepsy is influencing the present train of symptoms. It is certainly somewhat unusual for convulsions to occur at this early date after the injury. Convulsions following skull injuries occur usually either immediately after the accident, that is, within the first few hours, or they do not come on for months or even years. When they occur within the first few hours, they may either continue and increase until the patient dies, or they may gradually diminish and disappear. On the other hand, when they do not occur for months or even years after the injury, they may subsequently become very frequent, and the epilepsy ultimately terminate fatally. Many of you may remember the very able paper on this subject by the late Dudley Allen, of Cleveland. He reported a number of these cases and called particular attention to their course.

Remember that the early symptoms of intracranial hemorrhage are often irritative and the later symptoms paralytic. Not infrequently as a hemorrhage begins the patient develops spasms or convulsions, and as it continues and the important motor centers are compressed to the point of ischemia, paralysis ensues.

According to my experience, irritative symptoms are more frequent with subdural and cortical hemorrhages, and less frequent with extradural and subcortical hemorrhages. Careful hemostasis is very essential after intracranial operations, because if a subdural hemorrhage take place under the osteoplastic flap, the patient may have one or several convulsions as it develops, and sometimes these convulsions may continue, become almost consecutive, and go on to a fatal termination. Never allow blood to accumulate beneath an osteoplastic flap! Always drain—at least for twenty-four hours—unless hemostasis be complete!

I am plotting out the Rolandic fissure on the surface of the scalp to serve as an additional landmark for the flap. Here is the occipital protuberance behind, and here the glabella before. I determine the point half-way between the two, and then come a centimeter or a centimeter and a half forward. Now I am marking a line downward and backward, at an angle of about 60 degrees with the midline of the head. This constitutes approximately the line of the Rolandic fissure (Fig. 170). In operations upon the skull, I no longer, as a rule, pay any great attention to exact cranial topography. In the days of the trephine, when our openings were very small, accurate cranial topographic determinations were necessary, but now that we are using the large osteoplastic flap exclusively, we seldom have difficulty in arriving at the cortical focus which we are seeking. Furthermore, I believe that the trained eye of the surgeon who is constantly doing intracranial work becomes as accurate for all practical purposes as any craniometer on the market.

I am making the osteoplastic flap about the depressed fracture as a center. The bleeding from this flap as I turn it up is very slight, because, evidently, the blood-supply is interfered with by the cicatricial tissue beneath. . . . We are now down to the bone. We have about four electrically propelled instruments which we have used at different times for cutting through the cranium. When they work, they work well, but none of them works every time without fail. They all require constant and expert attention, and a second one must always be kept in reserve. We never start a cranial operation without having at least two of them sterilized and ready. The instrument which we have used most for many years was invented by Dr. Neff, my former associate. Of all that we have tried, it has given us the most satisfaction. We have recently secured a very expensive imported French instrument, but I did not have the courage to try it to-day. I must try it first on the cadaver, and then, before using it on a skull, I shall also try it in a resection of the tibia or in removing pieces of bone for transplantation.

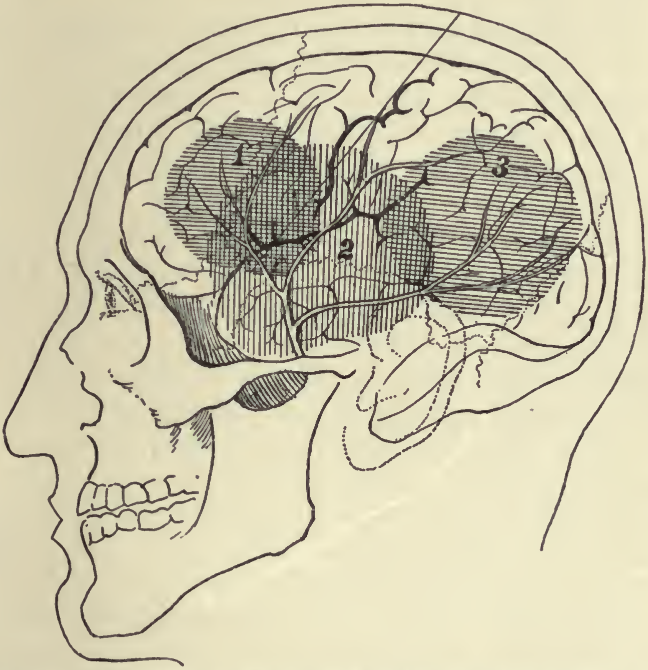

Fig. 170.—The location of hematomas of middle meningeal origin: 1, Frontotemporal hematoma, arising from the anterior branch of the middle meningeal; 2, temporoparietal hematoma, arising from either anterior or posterior branch or the main trunk; 3, parieto-occipital hematoma, arising from the posterior branch. The oblique line indicates the craniometric guide to the Rolandic fissure, as plotted by Dr. Murphy. Note the relative freedom of the leg center, which lies near the top of the central (Rolandic) area, from involvement in the hematoma, especially primary involvement.

This is Dr. Neff's trephine, with Dr. Zapffe's saws, which I am using now. To-day it seems to be working very well. . . .

We have outlined the osteoplastic flap, and it still remains to be broken through at the base. I use the small artist's chisel to nick it here before prying it up. . . . There is the middle meningeal artery bleeding now that I have broken back the flap. You can now see what happens when a trauma in this portion of the skull ruptures the middle meningeal artery. The artery bleeds most frequently, just as here, at the spot where it enters the bone. This is where it is most often ruptured. When ruptured during its course through the bone, or at the point where it leaves the bone, the ensuing hemorrhage is usually epidural. When, however, the rupture occurs where the branches of the artery run within the dura, the ensuing hemorrhage may be either epidural or subdural. Sometimes the artery is ruptured where it comes through the foramen spinosum. Such a hemorrhage is usually very severe and extremely difficult, in fact, practically impossible, to reach locally.

Remember that not only may the artery be ruptured, but also the two venæ comites. Also, the accompanying veins may be ruptured without the artery itself suffering damage. The arterial lesion, however, is the more frequent. Remember, too, that the cranium need not be fractured in order to produce a rupture of the meningeal artery. There have been a number of cases reported where this rupture has taken place without fracture of the cranium itself. The rupture of the vessels does not always take place on the side of the trauma, but sometimes occurs on the opposite side of the skull by *contrecoup*. Cases have been reported in which a rupture of the meningeal artery has occurred on both sides of the skull—on one side by direct injury and on the opposite side by *contrecoup*.

You can readily see how short a time it would require for the hemorrhage from that small artery to produce great intracranial pressure if the blood had no exit to the outside of the skull. This is the curved needle of von Eiselsberg which I am using to pass around the artery previous to ligation. I am passing it through the dura clear to the brain, just back of the site of rupture, and entirely around the artery. Do not try to grasp such a bleeding meningeal artery with the forceps. Merely pass a ligature around it on a needle, and then tie it from beneath, including the dura in the grasp of the ligature. Now the ligature is ready to tie. You see that the vessel is still bleeding as I start drawing the knot together. Now the knot is firm and the bleeding has stopped. That is the way I tie it. If you try to catch it with the forceps first, you will have to tie it, and retie it, again and again, because the ligature fails to reach the seat of the hemorrhage or slips off almost at once.

Let me cite a case just to show how rapidly such a hemorrhage may produce a cerebral compression which is fatal. A young polo player was struck upon the side of the head by the ball. The blow knocked him off his horse. He was unconscious only momentarily; then he regained consciousness and asked that the game be continued and tried to remount his horse, but his friends persuaded him not to do so. They took him home, he went to his room and to bed. There his friends left him for a few minutes, and on returning found him again unconscious. He was taken immediately to the hospital and operated upon soon afterward. The rapid onset of the second period of coma was a grave prognostic symptom. An enormous clot was found and evacuated, the hemorrhage was located and stopped, but the patient became worse and died. Why? Because the pressure of this enormous hematoma had shut off the blood-supply to the brain for so long a time that the cerebral cells were unable to recuperate from the prolonged ischemia. Remember that complete ischemia of the brain when of longer duration than twelve minutes is invariably fatal, and that partial ischemia when prolonged is always a serious matter. Could the unfortunate outcome in this young man's case have been prevented? If so, how? The patient could have been saved, I believe, if there were an immediately available, simple, and well-recognized treatment for these sudden and severe meningeal hemorrhages; and *there is*. The patient had to wait three hours after the accident before the surgeon started to operate upon his cranium. That is shorter than the average length of time which it takes such a case to reach the hospital, have a surgeon called, a diagnosis made, and the patient and the operator prepared for the surgical procedure.

For fifteen years I have advocated and taught the following method, which I think should have general recognition in the literature and become a standard practice. If this method were followed, it would take not over five or ten minutes for the surgeon to stop the hemorrhage when he has made the diagnosis. There is no occasion to wait three hours until the brain has become compressed to the point of fatal ischemia. All one has to do is to remember the anatomy of the bleeding artery. From what trunk does the middle meningeal come? It is a division of the internal maxillary artery. The internal maxillary in turn arises from the external carotid. The middle meningeal, after it springs from the internal maxillary, passes upward through the foramen spinosum into the skull. *Therefore, all that one needs in the management of such a hemorrhage, that is, all that is necessary for its prompt control, is to locate and tie the external carotid artery, which supplies the bleeding middle meningeal.*

The patient is unconscious—you need no anesthetic. Wipe a little iodin on the skin, burn off your instruments with alcohol, and then use them by the "no-hand-contact" method. Cut through the skin, expose the external carotid at the cornu of the hyoid bone, and just below the point where the digastric muscle crosses it, and tie it. You can do it in the barn, you can do it in the open, you can do it wherever you first see the patient, if you have your pocket-set with you. The ligation of the external carotid is not a serious proposition. The ligation of the internal carotid is serious, because this vessel supplies most of the arterial blood to the brain; but the regions supplied by the external carotid all have ample collateral circulation or consist of tissues which do not suffer severely from partial ischemia. The middle meningeal artery, however, is an end artery, and when its direct source of supply is shut off, it does not bleed from a collateral supply.

We have stopped the hemorrhage and are now examining the dura. As I look at it, I find it markedly thickened. Normally, one can often see the brain faintly through the dura; but I cannot see the convolutions through this dura. Neither can I see any dark blood beneath it shining through. . . . We have not seen any evidence of skull fracture yet at this site—no linear cleft still open, and no linear callus. I am turning back a dural flap which is pedicled in the opposite direction from the pedicle of my osteoplastic flap. Therefore, the two flaps will overlap when I close the wound and protect the brain better against subsequent danger of prolapse than superposed flaps will. Note as I turn the dura back how thick and opaque it is.

I have stripped back the dura and the underlying pathology is now in sight. We have here two cysts, probably of hemorrhagic origin. I am letting the fluid out of one. It is clear serous fluid. Now you can see the cyst sinking in as I evacuate it. . . . I am treating the second cyst similarly.

VISITING DOCTOR: Why do you say these are hemorrhagic cysts? The fluid which comes out looks perfectly clear.

DR. MURPHY: I have not only the history of this case to go on, but of many similar cases. Sometimes you find a cyst with blood still in it, and another cyst with only blood-tinged serum; sometimes a cyst with blood-tinged serum and another cyst containing only clear fluid will have the blood-pigment on its wall. There seems to be a marked tendency in the case of hemorrhage for the hemoglobin to become absorbed, precipitated, or deposited, before the serum is. This is true of hemorrhage not only in the brain, but also in the spinal cord. Why hemorrhagic cysts have all the hemoglobin absorbed finally and nothing left in them but the serum I do not know; but this is, I believe, clinically a well-established observation, and it is for the pathologist to explain *why* it occurs. In this second cyst the fluid seems to be slightly blood-tinged still. . . . The cysts are now both collapsed and their lumina obliterated. I am sewing the dura back into position, using just a few tacking sutures. The hemorrhage is now all stopped. I am replacing the osteoplastic flap and am suturing the skin. We shall dress this case just as we dress a case after decompression. I am placing no drain beneath the dura, because I have effected complete hemostasis there.

It is interesting to see how rapidly the absorption of the hemoglobin may take place in such cysts. Remember that it is only a month to-day since the patient's injury occurred, and already the hemoglobin has been completely absorbed from one cyst, and almost completely from the other.

I shall slip a narrow strip of gutta-percha tissue across the extradural field for the purpose of draining away any blood which may ooze from the scalp vessels. This small drain we shall remove at the end of twenty-four hours. For the scalp stitches I am using fine silkworm-gut. I am putting them in as interrupted sutures, and they furnish very satisfactory hemostasis of the scalp.

How beautifully the pathologic findings in the case fitted in with the history and with Dr. Mix's clinical conclusions! I must congratulate Dr. Mix on his accurate diagnosis. It is a great satisfaction to do surgery on cases properly diagnosed, for then one finds what one goes after. The patient's initial coma was evidently due to concussion. She came to hospital with a traumatic delirium two hours after the accident. The vomiting on admission was due to shock and not to increased intracranial pressure. However, a subdural hematoma slowly developed from the rupture of one of the tiny vessels—either a branch of the middle meningeal in the dura or a tiny pial vessel. Therefore, the patient, instead of coming out of the stupor due to concussion, still continued semicomatose, owing to the presence of the subdural hematoma. That she did not have an increased intracranial blood-pressure was due partly to the fact that the hematoma remained small in amount and became self-limited, and also to the fact that the existing shock still further lowered the blood-pressure.

At the end of three days the collection formed by the hematoma and the serous exudate was large enough to produce focal symptoms: that is, to produce the twitchings of the opposite side of the body recorded by the nurse and Dr. Kelly in the history. At about this same time the vascular oozing slowed up and stopped as the intracranial came to equal the intravascular pressure and clotting occurred in the ruptured vessel. Therefore, the irritative symptoms did not run over into a paralysis. Probably a preëxistent hyperirritability of the brain was an added factor in producing these spasms. Not only the small hematoma, but also the cyst resulting from it, produced the required degree of irritation, slight though it was, to start convulsive seizures in the already hypersensitive cerebrum.

I did not finish speaking about the immediate management of injuries of the middle meningeal artery. After one has tied the external carotid artery, whatever clots have already accumulated outside or inside the dura will necessarily remain there. But they are not liable to do any further harm than has already been inflicted, and they can be removed by craniotomy at a later period, if necessary, when the patient has been transferred to a hospital. Whatever pressure exists already, due to the hematoma, is not removed by ligation of the external carotid; but any continued increase in the pressure is thus prevented. That is the important point. That is the life-saving part of the procedure. It is the time which is lost between the making of the diagnosis and the beginning of the operation which determines the fatal outcome for the patient.

Ligation of the external carotid, remember, is not a treatment for *every* intracranial hemorrhage. It is the treatment—the proper emergency treatment—for severe hemorrhage from the middle meningeal artery. The most important point to remember is that you must make the diagnosis before you institute the treatment. Tying the external carotid will do no good if the patient is suffering from a hemorrhage from a pial vein. Neither will it do any good if the patient is suffering from fat embolism of the vessels of the brain, such as occurs after multiple fractures, particularly fractures of the pelvis and the big medullated bones. Remember that such a fat embolism may cause cortical symptoms not only closely resembling those of pressure on the motor region, but also coming on rapidly just as do the symptoms of middle meningeal hemorrhage. Remember, too, that fractures of bones elsewhere in the body are very frequent with fractures of the skull, and that one might have fat embolism and a middle meningeal hemorrhage occurring simultaneously. The importance of making the differential diagnosis is that one can secure splendid therapeutic results in middle meningeal hemorrhage by tying the external carotid promptly, whereas for fat embolism there is practically no remedy available if one excepts Wilms' rather dubious operative procedure, the draining of the thoracic duct—a very severe operation, of more theoretic than practical value.

Quite a useful diagnostic point to remember in this connection is the fact that in middle meningeal hemorrhage the convulsions never commence in, and never are confined solely to, the lower extremity. Neither is paralysis ever first noted in a leg or confined to it. The reason for this focal exemption is that the cortical center for the lower extremity is located high up in the Rolandic area, where the branches of the middle meningeal are extremely small and therefore very rarely rupture. Hemorrhages from the trunk or the larger branches of the middle meningeal may extend, and usually do extend, as far as the leg center; but the hemorrhages never arise primarily in the leg center—they occur there only secondarily, by spreading from the primary bleeding focus.

VISITING DOCTOR: What are you going to do for the patient's elbow?

DR. MURPHY: For her elbow we shall not do anything until the suppuration and the ossifying processes which you can see around the elbow in the *x*-ray plate have subsided. We shall secure such reposition and immobilization as is possible, but shall not think of undertaking any operative procedure for a long time yet. The elbow problem is a mechanical one. We have no fear as to the ultimate outcome there. Epilepsy, however, no matter what the variety, is not so easily reckoned with, and is the prime consideration with this patient.

[Following the operation the patient had no return of the convulsions. Her headaches disappeared, and she made a rapid and complete recovery.]

EXTRADURAL CEREBRAL COMPRESSION

Summary: Clinical history of patient; operation; living pathology of lesion; epileptic fit induced by etherization; uneventful recovery, with cure of symptoms.

HISTORY

The patient, a male aged thirty years, was admitted to hospital October 2, 1909. The family history is negative.

At the age of twelve years he dislocated the left shoulder in a fall from a barn window. Two years later he sprained the right ankle. Both injuries healed within the usual time. Eighteen months ago he had an attack of appendicitis, which was treated by the starvation method. He has never been jaundiced nor has he ever vomited blood. He occasionally passes blood in the stools after being constipated: this blood is due to hemorrhoids. He has never passed a stone in the urine or in the feces. He never had bladder or kidney disease.

The present trouble began about ten years ago, when, in a drunken brawl, he was hit upon the skull by a spittoon, which was thrown from behind. He fell on the spot unconscious, and did not recover consciousness until the next morning, when he found a scalp wound, three inches in length, on the right side. This was sutured and dressed, and he went to his business with no further symptoms. His physician told him that the outer table of the skull had been fractured. He had no convulsions, no epileptic fits—in fact, no irritations of any kind have so far manifested themselves in the motor zone. He complains now of impulsive desires to seek excitement in excesses, especially when under the influence of strong drink, in gambling, racing, automobile speeding, etc., and also of a loss of stereognostic sense, so that he changes objects from right to left, as on the right they feel lighter.

OPERATION

[Performed by Dr. Murphy October 4, 1909.] On freeing the periosteum there was found a pronounced depression, oval in shape, just behind the stellate point of the skull, which showed evidences of a previous hematoma. The bone was eburnated and differed from the neighboring bone. There was no diploë. The pathologic condition showed that the bone had been fractured, and that it was much thicker than normal. The dura was infiltrated, injected, and mottled. A piece of dura the size of a half-dollar was excised. There was no evidence of a previous leptomeningitis, although the sulci were deeper and there was more fluid than normal. There were no adhesions to the subdural layers. The arachnoid and pia were not adherent to each other nor to the brain. The osteoplastic flap was replaced and secured with periosteal sutures, and finally with scalp sutures. The flap was replaced to sustain pressure. A portion of the dura was freed from the bone ¾ inch around the entire zone, and from the flap 2½ by 3 inches.

On starting the anesthetic the patient developed an epileptic fit, and following the fit there was a general body tremor.

The patient made an uneventful recovery. There was a primary union of the flap on October 12, 1909, eight days after the operation. He left the hospital on November 6, 1909, in good condition.

FEBRUARY 1, 1915: The patient's wife returns to-day saying that her husband is completely cured. He has had no more convulsions, headaches, nor the slightest inclination to alcoholism, and he is a business man of the most sterling type. This shows that, notwithstanding the slight changes that had taken place in the bone and the even slighter change in the dura, the operation has completely cured the condition.

ANCIENT FRACTURE OF SKULL—OSTEOPLASTIC EXPOSURE—FASCIA-FAT TRANSPLANT INTO DURA

Summary: Clinical history of patient; comments; convulsions inevitable without operation—easy to prevent, but difficult to cure by operation; technic of operation; severe headaches cured by resection of dura—interposition of fascia-fat transplant; discussion of clinical course of head injuries; management of head injuries when seen early; citation of illustrative cases; tapping of ventricle to relieve convulsions; importance of removing dura in addition to bone; percentage relieved by treatment; slight risk of operation; convulsions at times fatal.

HISTORY

The patient, a male aged forty-eight years, was admitted to hospital November 17, 1914. The past and venereal histories are negative.

The present trouble began two and one-half years ago, on March 12, 1912, when the patient was struck upon the head over the frontal region by a broken cable. He was knocked down and was unconscious for five minutes. Then he got up and walked to hospital. He had no other attacks of unconsciousness, nor was there any bleeding from the ears, nose, or throat. There was a fracture of the frontal sinus. He left the hospital on March 27, 1912, fifteen days after admission. Two days later, on March 29, 1912, he developed severe frontal headaches, vertigo, and spots before the eyes. This condition continued. He was not relieved by medical measures. Seventeen months after the injury, in August, 1913, decompression was performed. The frontal headaches still continue. He has had no convulsions.

COMMENTS AND OPERATION

Dr. Murphy: This is a classic case—classic in the increase in the frequency of the periodic headaches, consummating in convulsions. Spasms may occur early, as well, in the course of the trouble. This man had the central symptoms early—very much earlier than the average patient. He is now having severe headaches that are incapacitating him. We know it will be but a short time until he has convulsions. We know convulsions are headed off in a large percentage of cases when operated upon before the convulsion stage. We know further that cures are obtained with difficulty after the convulsions have started. We want to make an osteoplastic flap and to remove a considerable area of the dura, but are handicapped by the previous incision into the skull. We can secure partial hemostasis by adjusting this compressor down to the periosteum and then pushing it well across the field. It is not completely hemostatic, but it at least lessens the quantity of blood that escapes.

The patient was unconscious but a brief time. There is the cavity made from the previous operation: we want to get behind this depressed area. Now we detach the dura from the bone, beginning at the margin. Here is some fibrous tissue: there, a spiculum of bone. This is the instrument we used before we had the electric machine—the de Vilbiss. We have also a very good machine, devised by Dr. Neff, which facilitates work considerably, but it broke the other day. In all this work we like to make a large flap: now we have outlined the U. We follow the line of fracture by elevating the tissues subperiosteally. The dura is now free and an area three inches in diameter is excised. We next cover this developed field over with sponges, and turn our attention to the thigh, from which we shall obtain the fascia-fat transplant. [Flap of fascia lata and fat removed from thigh and inserted into the dural defect.] Hemostasis is absolutely necessary in all this work, for it saves trouble. In the dura and pia it may best be obtained by the needle and thread. The oozing is from the capillary vessels in those two membranes. This flap needs but a couple of tacking stitches, and a silkworm-gut drain to prevent hematoma formation beneath the flap, which would be a serious matter; the drain is removed at the end of twenty-four hours. Now the scalp falls back into position and is fixed with horsehair sutures.

Let the record show that there was a spiculum of bone sticking into the dura under the upper margin of the site from which the fragment had been removed; that the dura was adherent to the scalp over the area exposed by removal of the flap; that a flap of fascia lata and fat was taken from the thigh and inserted over the area from which the dura had been removed; that the flap was tacked into place, and that drainage was provided for twenty-four hours.

That is the regular course of traumas to the head. First come the recurrent headaches; second, there comes the *petit mal* or a typical epileptic convulsion, and then increase in frequency of the epileptic attacks.

Now let me give you the clinical course of some of these cases, that you may the more fully appreciate the importance of head injuries. I have used this expression many times: I would much prefer to be killed than to have an accident to the skull, associated with any type of fracture that renders me unconscious, because such a large percentage of these cases results in convulsions later and finally in mental disturbance. There is no limit to the time that may elapse between the injury and the onset of convulsions. Illustrative of this statement, I cite a case that occurred in my early practice on Newberry Avenue, near Eighteenth Street: A child, two years old, fell from a second-story window, striking the pavement with some force upon the vault of the skull, and was almost dead when I arrived. He had a comminuted fracture of the skull, the fragments beneath feeling like a bag of bones. On palpation the calvarium seemed to move *en masse*. After a period of unconsciousness which lasted three weeks, he finally recovered and grew up as an irritable but otherwise natural child. He graduated from the high school, took an extra two-year course, and then entered a bank. With the exception of recurrent headaches, things went along nicely until twenty-two years after the accident, when suddenly, one day, he had a convulsion in his office. Convulsions recurred every two or three weeks without failing. At operation we removed a considerable portion of the skull at the site of depression. Nothing showed there except a little more firmly adherent dura: there was no spiculum of bone—no other source of irritation. He recovered from the operation without an untoward symptom, and then went on until he had the next convulsion, about four months after the operation. The convulsions have continued with increasing frequency until at last his mind is deteriorating, and eventually he must enter an asylum.

I recall a similar case, seen twenty-seven years after the accident—one which I did not treat originally. The fact that such irritations can and do occur after these injuries means what? That when we have these cases to treat primarily we should exercise extreme caution in what we do. The patient who has an accident rendering him unconscious from a blow upon the skull should not be permitted to leave his bed for a period of six weeks, no matter how he feels or what he wants to do. From observations upon a number of cases we believe—though it savor of an empirical statement merely to say it from clinical experience—that the patients whom we have kept in bed for a long time with an ice-bag applied to the skull have had the least trouble subsequently—the fewest headaches and the smallest number of convulsions. That belief is difficult to support with statistics, but it is the clinical estimate. Statistics have little or no value in this class of cases; many have convulsions, however, and therefore in later years our management has been to place them in bed at once. At the end of four or five days, unless the patient be unconscious, if there is a depressed fracture, elevate it; if it is compound and comminuted, remove the loose fragments; if not compound, elevate the bone flush, so as to leave no gap in the skull. Even after primary healing of the wound insist upon the patient's remaining in bed. If he has had no wound at all, again insist that he remain in bed. That plan of treatment did not originate with us: it was the plan carried out by the late Dr. Fenger, whose statements correspond with our clinical studies. He had a large and extensive experience in this type of work, from which he drew his own conclusions.

Since we do not know the etiologic pathologic change in this or in any other given case of epileptic convulsion, we must treat empirically, upon the plan that gives the best results. The severity of the concussion bears some relation to the final result as regards convulsions: the blunter the instrument, the severer the concussion.

That the removal of the dura mater inhibits the convulsions is the consensus of opinion in the profession at the present time. Formerly a portion of the bone alone was removed at the site of the compound fracture or of the depressed fracture, but without good results. We cite a case illustrating the beneficial effect of the removal of the dura. A boy was hurt in a runaway accident in 1888. I remember the year, because it was ten years before the Spanish-American war. He was riding in a sleigh, the horse bolted, he was thrown out, and his head struck against a telegraph pole. Following this accident he had headaches, which became more and more frequent, and finally convulsions appeared repeatedly in 1898, ten years after the accident. He came here to Chicago and we elevated a large osteoplastic flap, just as you saw me do to-day. We also removed a small fragment of bone, to which we did not, however, attribute any special significance as an etiologic factor in the convulsions, but thought that the elevation of a flap of dura would benefit him. He went home, and in six weeks very severe convulsions began—as many as five or six in a single day. My associate at that time, Dr. Lemke, went to see him. Dr. Lemke was proficient in anatomy, and had had a large asylum experience with this class of cases before coming to us. He passed an ordinary exploring needle through the seam in the skull where we had trephined, punctured the lateral ventricle, and allowed the fluid to escape. The convulsions subsided forthwith. The patient returned to Chicago the next week. We reopened the skull and excised the dura extensively, just as we did this morning, except for the interposition of fascia and fat. He had no further convulsions until last February—a period of sixteen years. In February a fleeting convulsion occurred, and was followed a week or two later by a typical convulsion. These attacks have continued. Now you see why we attribute the freedom from convulsions to the removal of the dura: it is because, after the previous operation, at which only a small fragment of bone was removed, he had convulsions as severe as, if not severer than, the previous ones. He is now beginning to show a little mental disturbance. He was established in the real estate business here in Chicago, and had made quite a little fortune and reputation for himself in the interim.

I cite these cases merely to show you the results we have obtained, but if you ask me what percentage of these cases is relieved by operation, my answer is, 5 per cent.—one in twenty. This is a disheartening state of affairs, but at the same time you must remember that every one you rescue by an operation goes to your credit account, and that you have exposed the patient to practically no operative risk, because the operations are not fraught with danger. In patients having convulsions, however, decompression with removal of a dural flap is not entirely without danger. I have seen severe, and even fatal, convulsions the day after operation. Why rapidly fatal convulsions occur once in a while, though rarely, I do not know, but the clinical fact remains.

TUBERCULOUS MENINGITIS

Summary: Clinical history of patient; discussion by Dr. Mix; the finding of tubercle bacilli in the cerebrospinal fluid; rarity of convulsions in this type.

HISTORY

The patient, a female aged eighteen years, was brought to hospital January 31, 1915, and recited the following history:

She was taken sick two weeks previously, on January 16, 1915, and was under a physician's care five days,—until January 21st,—when she was taken to her own physician. At this time her face was flushed from fever, she was irritable, her lips were dry, and her tongue was coated. Her temperature ranged from 100° to 102° F., her pulse from 100 to 112, and varied in character. Her blood-pressure, taken with the Tycos instrument, was 118 to 122. Respirations ranged from 22 to 24. Two Widal tests, made before admission to hospital—one on January 25th and the other on January 30th—were negative. The bowels moved normally until January 31st, when rectal control was lost. Urinalysis showed specific gravity of 1017, increase of phosphates, slight increase in urea content, no sugar, no albumin, no indican, and negative diazo-reaction. The abdomen was tympanitic, and she complained of occasional pain in the right iliac fossa. The abdominal muscles contracted at times. She had persistent frontal headache. Examination of the lungs revealed areas of dulness, which disappeared later, and inconstant râles of varying character.

Dr. Murphy: Dr. Mix will consider this very interesting case.

Dr. Mix: The patient is a girl about seventeen years of age, who on the sixteenth day of January was taken ill with what was diagnosed as bronchitis—a not infrequent diagnosis for tuberculous meningitis.

When I saw the patient she presented the picture of meningitis. The pulse, at first slow, now ranged from 110 to 114; her temperature varied from 101° to 103° F. She was stuporose. When asked to protrude the tongue, it stayed out. Her mother said she had ptosis of the left eye two or three days previously. While watching her an occasional muscular twitch appeared—one eye twitched but the other did not.

The cerebrospinal fluid obtained by lumbar puncture showed 12,000 cells to the cubic millimeter and a few tubercle bacilli. The finding of tubercle bacilli is most unusual. We have carefully examined the cerebrospinal fluid in several cases of tuberculous cerebrospinal meningitis. For example, during the past year I have seen six cases which have been followed out by inoculation of the cerebrospinal fluid into guinea-pigs, but no tubercle bacilli could be found at all. This is the first case we know of that has shown them.

The quantity of cerebrospinal fluid that must be removed if tubercle bacilli are to be found is considerable, because one must have a large amount to centrifuge. The larger the amount of fluid centrifuged, the more cells and the more bacilli present.

In tuberculous meningitis the chief pathologic process locates usually at the fissure of Sylvius and the fissure of Rolando, on the outer surface of the brain. Infrequently it may happen that tubercle bacilli escape from the tubercles and assemble in the cerebrospinal fluid; more frequently, however, they remain in the tissues. In a case of this sort there may be tuberculosis of the ependyma, since at postmortem examination the ependyma occasionally reveals marked evidence of the tuberculous process.

This slide shows the tubercle bacilli perfectly clearly and taking the characteristic stain. There is no chance of error, because the technic of finding these tubercle bacilli was faultless. Tubercle bacilli have been reported in certain instances because of error in mistaking for them acid-fast bacilli from the distilled water, in which the latter appear as a contamination. In this case the chance for such an error was impossible, because the cerebrospinal fluid was collected in a sterile test-tube through a sterile needle, centrifuged in a sterile tube immediately, and examined upon a slide that had just been cleansed in alcohol. Distilled water came in contact with the slide during the washing only, and after the specimen had already been stained.

This patient had had no convulsions. In tuberculous meningitis convulsions are the exception. The only symptoms and signs usually observed are headache, nausea and vomiting, slow pulse, Kernig's sign, *tache cérébrale*, retraction of the head, and hypertonus of most muscles of the body. In the case of basilar meningitis eye signs are usually added. In tuberculous meningitis basilar symptoms are not so uncommon as in pneumococcic, streptococcic, staphylococcic, or diplococcic meningitis. Nevertheless in a tuberculous case third nerve signs, as dilatation and inequality of the pupils, and at times internal or external squint, are found occasionally. Once in a whole a sign suggestive of nystagmus is observed. Nystagmus is a slow pulling away of the eyeball with sudden return. In this case of meningitis, and in many others, one often notices a quick jerk away from the midline and a slow return of the eyeball. This phenomenon showed distinctly in this patient yesterday.

TUBERCULOUS LEPTOMENINGITIS AND EPENDYMITIS WITH NECROPSY

Summary: Clinical history of patient; discussion by Dr. Mix; steps by which diagnosis was reached; characteristic symptoms of tuberculous meningitis—disproportion between temperature and pulse; additional signs; terminal course of case; report of autopsy; report on brain by Professor Zeit; congenital megalocolon (Hirschsprung's disease) found; anatomic basis of the epilepsy; result of guinea-pig injection.

HISTORY

The patient is a male, aged nine years, who was admitted to hospital October 5, 1914, with the following history:

At full term he was delivered by instruments. The mother was in labor twenty-four hours. After delivery a depression just above and behind the child's left ear was present. No symptoms appeared until he was three months old, when there occurred an attack of frothing at the mouth, with rolling back of the eyeballs. He had two or three spasms in the two weeks following, was very nervous, and did not sleep well at night. At the age of seven months he had an attack of German measles, and was sick three or four days. At the age of eighteen months, while teething, he had a severe convulsion which lasted from 4 o'clock in the afternoon until 1 o'clock the following morning. During this period he was unconscious, and remained semicomatose the next three days. He was in bed two weeks.

He had no more attacks of this kind until he was two and one-half years of age. At that time his mother noticed that, while eating, he dropped his spoon, stared, and then lost consciousness momentarily. These attacks recurred every two or three days for three months. When three years of age he had attacks of unconsciousness for five or ten minutes, after which he slept heavily several hours. Before the onset of these attacks he clutched at the stomach and complained of pain, as an aura.

These attacks gradually increased in frequency, often occurring two or three times a day. This condition continued until summer, when he was sent to a farm. Here he seemed to improve, having only four or five attacks in five months. In the fall of that year the attacks increased in frequency and varied in severity. He had as many as 16 in one day.

When four years of age his parents took him to a specialist in cerebral surgery who said that the spasms did not warrant an exploratory operation at that time. He was given the bromid of calcium. He remained in Baltimore two months. He had one or two attacks a day during that time, most of which were violent, and of the *grand mal* type. This condition lasted until spring, when his parents took him to an internist who advised putting him in a private school for epileptic children. While in the school the attacks decreased in number.

Two months ago, in August, 1914, he had five attacks. Two weeks ago, on September 20, 1914, he had a severe attack, lasting from 6.30 to 8.30 P. M. Two days later, at 8.30 P. M., he had an attack of *grand mal* with paroxysmal headache confined to the right side. During the eight days preceding admission he had attacks of nausea with vomiting, fever of 102.8° F., slow, irregular pulse, profuse head sweats, and hypersensitiveness to light, touch, and sound. Four days previous to admission, on October 1, 1914, he became semicomatose. There was ptosis of both eyelids. The voice was reduced to a whisper. It was difficult to arouse him, and he rapidly became emaciated. On the day of admission he showed, in addition, marked rigidity of the neck.

Two years ago he was sick, but the nature of the trouble is unknown. One physician diagnosed it appendicitis and another, pneumonia. We are unable to elicit the symptoms of that malady.

There is no family history of epilepsy. Syphilis also is denied. His mother had one miscarriage before, and one after, he was born.

COMMENTS AND OPERATION

DR. MURPHY: This patient was brought to me in his last acute illness as a possible surgical case in the belief that he might have had a chronic cerebral abscess which had resulted in an acute cerebral infection. The case was therefore a border-line case, and, proving to be medical, was turned over to Dr. Mix. Dr. Mix has examined this patient and will take up the discussion.

Dr. Mix: The patient is a young school-boy nine years of age. His history is very interesting. You have heard that the birth followed a hard labor, the delivery having been instrumental. Furthermore, immediately after birth a depression was found above and behind the left ear. Whether there was any relation between this instrumental delivery, the depression, and the subsequent epileptic seizures is problematic. When the brain is injured by an instrumental delivery, it is usually difficult to induce the new-born baby to breathe, because the pressure of the subdural hemorrhage consequent upon the trauma from the instrument prevents normal respiratory function. Inquiry into the state of respiration following birth was fruitless, for, as the mother remembered, the boy breathed normally after birth.

The epileptic symptoms began early in life. At three months of age he was seen to froth at the mouth and to roll the eyeballs: two or three such spasms were observed. No importance was attached to them by the mother, however, until the child was eighteen months of age, when, while teething, he had a severe convulsion, lasting nine hours,—from 4 P.M. to 1 A.M.,—remained semicomatose the ensuing three days, and was confined to bed two weeks. Such a seizure is not an ordinary epileptic manifestation. A long-continued convulsion followed by a semicomatose condition of three days' duration is suggestive of a most serious vascular disturbance.

When two and one-half years of age, the patient's mother noticed attacks in which, while eating, the child lost consciousness momentarily, dropped his spoon, and stared. These attacks occurred every two or three days for about three months. Evidently they were attacks of true *petit mat.* When the child was three years of age, the epilepsy became much more marked. The attacks were longer, lasting from five to ten minutes, and were followed by deep sleep. Noteworthy is the fact that *the patient clutched at the stomach before the onset of the spasms, and* *that he complained of pain just previous to the attack.* This suggests some relation between an abdominal irritation and the attacks of epilepsy. The epileptic seizures gradually increased in frequency to two or three a day, although they often intermitted for a week at a time. They occurred so frequently that, at the age of four years, operation was considered seriously. None was performed, however. The patient was given the bromid of calcium, which modified the attacks markedly.

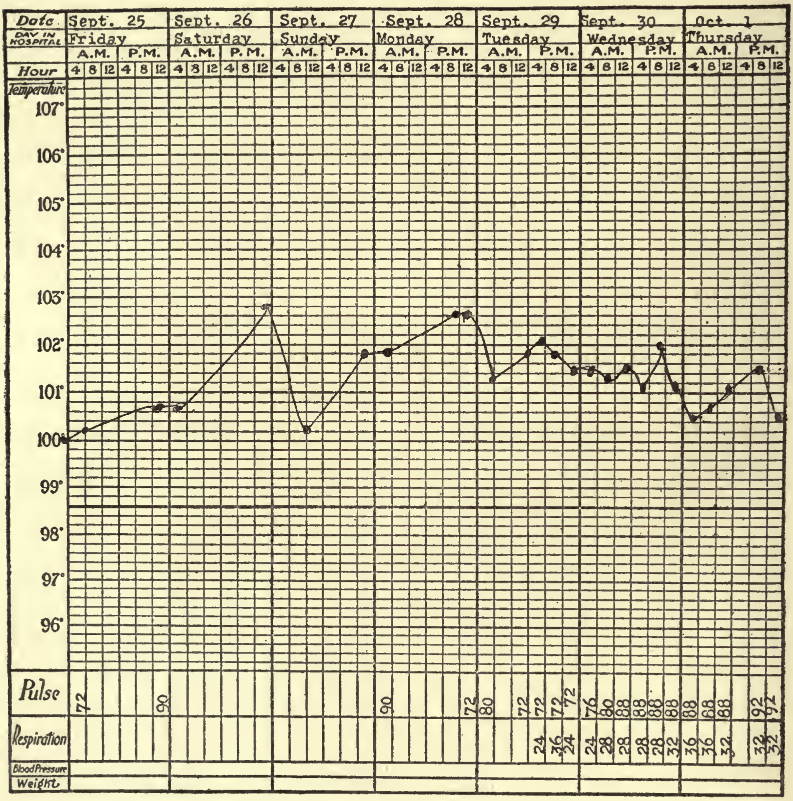

Fig. 171.—Temperature chart, first week.

The attacks, however, have never ceased. From the age of four years, until last year, they were numerous. It was advised that the boy be placed in a school for epileptic children, and this was done.

While at school the boy developed another illness. We tried to obtain a complete account of the condition from the time of the onset of the sickness until the fifth of October, which was the first time we saw the patient. We learned that on September 20th, between 6.30 and 8.30 in the evening, he had a severe convulsion. Two days later, on the evening of September 22d, he had another convulsion, also of great severity. This was unlike the other convulsions in the addition of a severe headache involving the right side.

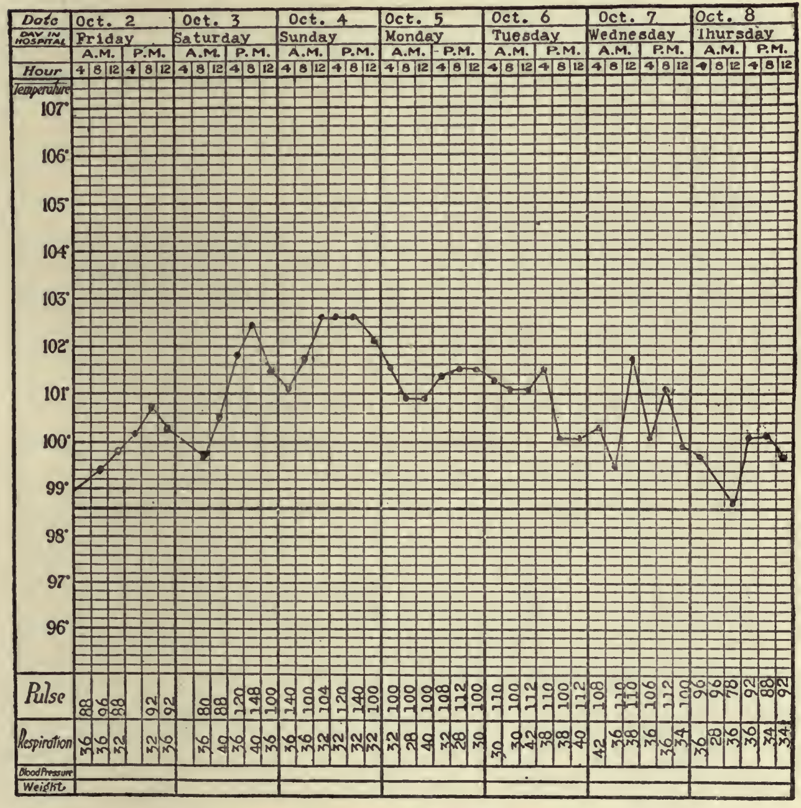

Fig. 172.—Temperature chart, second week.

On September 25th the school physician observed that the patient had fever. His temperature, taken twice on that day, ranged between 100° and 101° F., and the next night it was 102.8° F., which equaled that previous to admission into hospital. His pulse varied from 72 to 90. On the evening of September 28th it was observed that, although the temperature had reached 102.5° F., the pulse was but 72. Furthermore, on September 27th, the day before this, the physician found the pulse slow and irregular. He also noticed profuse sweating about the head, hypersensitiveness to light, resentment at being touched, and that all noises were disagreeable to the patient.

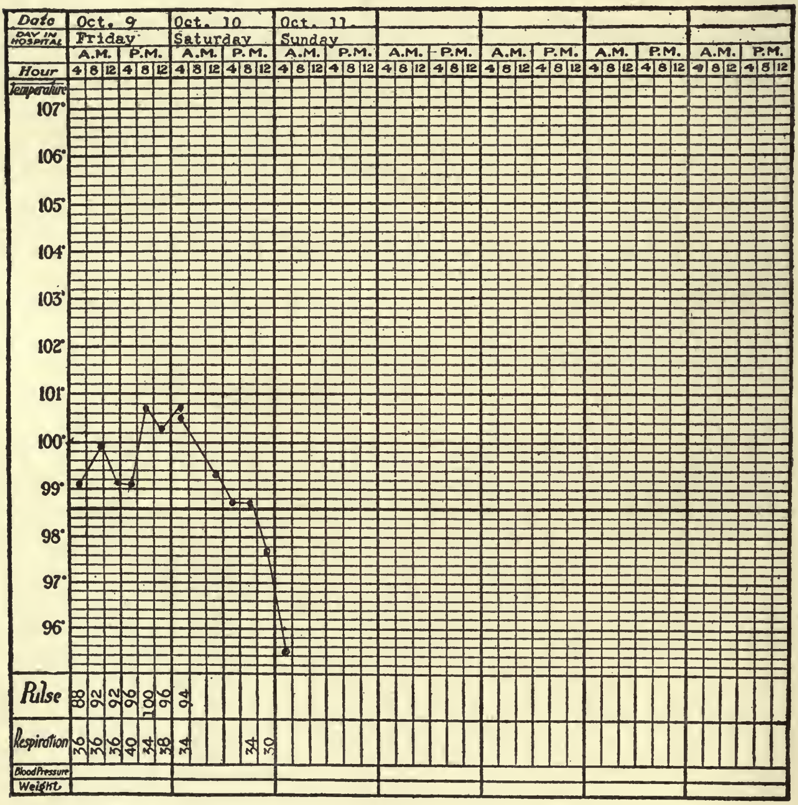

Fig. 173.—Temperature chart, closing days.

His condition remained stationary throughout the twenty-eighth, twenty-ninth, and thirtieth of September, the pulse increasing gradually in rapidity from 72 to 88 beats a minute, and the temperature averaging 101.5° F. On October 1st he became semicomatose for the first time, and showed a sign of disturbance of the eye muscles in ptosis of both eyelids. The voice was reduced to a whisper. On October 5th he was brought by train to Mercy Hospital, where we saw him immediately after admission.

The blood examination showed 14,000 leukocytes. The urine contained no albumin or sugar, no casts or red blood-cells; specific gravity, 1025. Although there were 14,000 leukocytes, yet a Widal test was made and, as was expected, was negative—in fact, the test was unnecessary. We have never seen the leukocyte count above normal in typhoid fever except in the presence of a perforation with peritoneal involvement or with some metastatic focus of infection.

I first saw the patient on October 5th at noon. He was in bed with the eyes closed, apparently asleep, but it was not difficult to arouse him and to get him to answer questions. We learned that he had had frequent headaches; that he had vomited once, although the vomiting was not of the projectile type; that he had been constipated; had no desire whatever for food; that he complained bitterly on being touched, and that he objected to light. His posture was characteristic. Both arms and legs were flexed. The legs could be straightened without eliciting evidence of pain, but he immediately flexed them again. There was moderate rigidity of the neck, with very slight retraction of the head. The retraction was minimal, but attempts to lift the patient by the head increased the rigidity of the neck. Kernig's sign was positive; the knee-jerks were much diminished; the cremasteric reflexes were present; the epigastric and abdominal reflexes much reduced, and there was no Babinski sign in either foot. Manipulation of the extremities caused pain, and there was hypertonus of all the muscles of the body. Indeed, the painful hypertonus was the most striking feature, it being responsible for the Kernig's sign, the rigidity of the neck, and the posture.

Examination of the head revealed disturbed balance in both eyes—a lateral but no vertical conjugate deviation. The pupils were unequal, the right being the larger; they reacted to light and to accommodation, and showed no paresis. The ptosis, mentioned by the mother, was present no longer. There was no disturbance involving the cranial nerves other than the third.

In view of these findings it was incumbent to perform lumbar puncture at once. The fluid was under considerable tension, so that 30 c.c. were withdrawn very rapidly. It was perfectly limpid—as clear as spring water. Each cubic millimeter contained 1360 cells, of which 22 per cent. were polymorphs, 76 per cent. small lymphocytes, and 2 per cent. large mononuclears.

Because of the characters of the cerebrospinal fluid, the diagnosis of tuberculous meningitis was established. In meningitis due to *Diplococcus intracellularis*, *Streptococcus*, *Staphylococcus*, or *Bacillus influenzæ*, the cerebrospinal fluid is constantly turbid: in tuberculous meningitis it is clear, although it may present a light, ground-glass appearance in the test-tube. However, cerebrospinal fluid containing but 22 per cent. of polymorphs and 76 per cent. of lymphocytes, when associated with fever, headache, nausea and vomiting, and slow, irregular pulse, can indicate hardly anything else than tuberculous meningitis.

The most characteristic symptoms of tuberculous meningitis are irregular fever, headache, nausea and vomiting, and slow, irregular pulse. The behavior of the pulse is characteristic. In the early stage it is disproportionately slow, in this case 72, although the temperature was 102.4° F. While a similar pulse-temperature relationship is seen in typhoid fever also, yet at the onset of typhoid fever headache is never associated with nausea and vomiting. This is extremely important as an early differential diagnostic point. Later on the pulse in meningitis increases in rate, so that by the second week it is commonly above 100. In tuberculous meningitis—indeed, in all forms of meningitis—the pulse increases to an astonishingly rapid rate just before death, and we therefore predict, that in the terminal stage of this boy's case, the pulse will increase to 160, or perhaps even more.

Besides these symptoms of irregular fever, headache, nausea and vomiting, and slow, irregular pulse, other later signs of tuberculous meningitis of extreme importance are observed. The first is hypertonus. This involves most muscles of the body; consequently attempts to extend the leg upon the thigh flexed upon the abdomen meet with instant resistance. This phenomenon constitutes Kernig's sign. Moreover, upon lifting the patient by the head the trunk is lifted simultaneously. This rigidity of the neck is also but a sign of hypertonus. Finally, a scaphoid abdomen is frequently observed, indicative both of hypertonus of the muscles of the anterior abdominal wall and of absorption of the mesenteric fat. It follows that the scaphoid abdomen is not observed in the early stages

Another important sign is the behavior of the third cranial nerves. Often there is ptosis of one or both eyelids, and paresis of one or both of the internal recti. Furthermore, pupillary disturbances are common, inequality being the rule. Irregularity of the pupil as a pure meningitic sign does not occur. *Hippus*, or oscillations of the pupil when exposed to light, is sometimes observed. Usually the pupillary inequalities are transitory—they are present one hour and absent the next. Dilatation of the pupils is also common during the early stages of cerebrospinal fever.

Of negative value in this case is the absence of herpes labialis, which is seldom found in tuberculous meningitis, although it is common both in the pneumococcic and in the diplococcic varieties.

More important than the clinical signs is the behavior of the cerebrospinal fluid. The tension is constantly, slightly, and at times materially, increased. It is usually, though not always, limpid. In the test-tube it rarely attains the degree of opacity that merits comparison with ground glass. Examination of the centrifuged specimen, which must be made immediately upon withdrawal, shows increase in the cell-count, in this case to 1360 per cubic millimeter. Differential count shows that the lymphocytes predominate, as in this case, where they formed 76 per cent.

General examination of the body revealed no sign of any other malady. The heart, lungs, liver, spleen, and kidneys were normal. One noteworthy fact in this patient is the absence of retraction of the anterior abdominal muscles, which is usually present. *Indeed, unlike most cases of tuberculous meningitis, this patient's abdomen protrudes and is tympanitic.* We cannot explain this exceptional degree of tympany, for tympany in meningitis is contrary to the rule, thus differing entirely from typhoid fever. We, therefore, diagnosed tuberculous meningitis, but have been unable to determine the original focus of infection.

After examination the day of admission, on October 5th, the patient became steadily worse. During the night of the fifth he was delirious and had a severe headache; the pulse became weak, and he slept about an hour only. On the sixth of October the temperature fell, but the pulse and respiratory rate both became more rapid. On the morning of the sixth twitching of the lips was observed. Although semicomatose during most of the forenoon, at intervals he aroused himself spontaneously. In the afternoon he perspired profusely and was delirious, picking at the bedclothes, much as a typhoid-fever patient in the third week. On the evening of the sixth there was difficulty in swallowing. Subsequently he fell into stupor, from which it was almost impossible to arouse him; during the night there was muttering delirium.

On the morning of October 7th the patient awoke rational. The respirations were rapid and shallow, but the fever was not high. He complained of pain in the stomach, and later in the forenoon of headache. On the afternoon of the seventh he became stuporose, and it was hard to arouse him for nourishment. During the late afternoon he was quiet, and in the early evening awake and rational, but unable to urinate. During the night of the seventh to the eight he was awake and rational at intervals, restless, and complained of severe headache. He muttered in his sleep. During the forenoon of the eighth the temperature at one time was normal and the pulse 78. He seemed rational when aroused, and perspired copiously about the head. At this time he urinated freely. On the evening of the eighth the hands trembled slightly, and he lost control of the bladder.

During the night of the eighth to the ninth he was awake several times and was rational.

About 4 o'clock on the morning of the ninth there was an involuntary passage of urine. On the afternoon of the ninth there was difficulty in swallowing, yet he could take nourishment. During the night of the ninth to the tenth he was awake and rational at intervals, but moaning when sleeping. Early the morning of the tenth Cheyne-Stokes breathing was noticed for the first time. This is always of bad prognostic significance. On the morning of the tenth he could be aroused at times, but was stuporose. Occasionally the breathing was irregular. At noontime on the tenth severe intermittent twitching of the muscles began and lasted five minutes at a time. That evening the same phenomenon was observed in the right arm, and the rigidity increased, although urine was passed three times on the tenth with his knowledge and control. On the evening of the tenth there were two involuntary bowel evacuations. Although after 3 P. M. on the tenth the temperature was normal, the pulse, which was 90 early in the evening and 100 at 9 P. M., became weak and intermittent.

At midnight there appeared a convulsion which lasted three minutes. This convulsion is noteworthy, since in tuberculous meningitis convulsions are rare, while in pneumococcic meningitis they never occur.

Early the morning of October 11th his body was covered with cold sweat, the temperature falling to 95.4° F. and the pulse remaining at 100, but at times it could not be counted. The twitching of the muscles became almost constant. During the forenoon, until his death at 12.03 on the eleventh, he was resting quietly, but the pulse was feeble; he was moaning, and at times slight twitching appeared in various parts of his body.

Note the following pulse changes, which are in accordance with the predictions made when this case was demonstrated. The pulse was 130 at 6 A. M., 130 at 8 A. M., 128 at 9 A. M., 144 at 10 A. M., 148 at 11 A. M., and just before death it could not be counted. So far as I have been able to observe, this is the rule, and it has been mentioned before in the CLINICS. One can always predict the exitus of a case of tuberculous meningitis by the acceleration of the pulse. In these cases death is never sudden, but approaches gradually, hour by hour. When the temperature falls and the pulse rises, death takes place within twelve hours' time.

Appended is the serial chart showing the range of temperature from the beginning of the illness until death (Figs. 171, 172, 173). (For the record from the twenty-fifth of September to the fifth of October we are indebted to the physician in charge previous to the patient's admission to Mercy Hospital.)

Autopsy, which I performed the afternoon of his death, revealed the following features: The brain was hardened and examined by Professor F. Robert Zeit, of Northwestern University Medical School, who reports thus: "*Leptomeningitis basilaris tuberculosa et ependymitis tuberculosa. Hydrocephalus internus.* The ependymal lining of the lateral ventricles, especially of the anterior horn of the left lateral ventricle, is studded with miliary tubercles. The pia mater of the base of the brain and along the Sylvian fissures, and particularly the pia mater over the left supramarginal gyrus and over the lower third of the left postcentral gyrus, shows numerous miliary tubercles. There is also a moderate degree of hydrocephalus internus." Professor Zeit found no trace of a solitary tubercle or tuberculoma to account for the tuberculous meningitis or for the epilepsy. At his death we suspected as the cause of the epilepsy a solitary tubercle, and that this tubercle had involved the meninges in general tuberculous meningitis; yet, after removing the brain with the greatest care, no solitary tubercle could be found.

The condition in the abdomen was most interesting. The colon was markedly dilated. The patient's mother felt that the boy's epileptic seizures had had some association with an abdominal condition, and requested that the colon be especially examined at autopsy. She felt that the colon was at fault, because when special attention was given to the child's bowels by flushing the colon every day by high enemas, the child was freest from convulsions: in fact, when the colon was lavaged vigorously for three months he had no convulsions. Congenital dilatation of the colon (Hirschsprung's disease) to two and one-half or three inches in diameter explained the enormous distention with gas, previous to and during his last illness. The cause of this dilatation was stenosis from a congenital band two inches above the rectosigmoid junction. It is possible that the Hirschsprung's disease, by reason of the absorption of toxins, might have had etiologic bearing upon the epilepsy, but I do not believe that it was the whole cause.

Furthermore, it is possible that the instrumental delivery might have been partially responsible for the epileptic seizures. This is more probable, as the forceps left an imprint above and behind the left ear. Although, according to the history, the child frothed at the mouth at the age of three months, yet the attack which occurred when he was one and one-half years old, and during which the convulsions lasted several hours and were followed by three days of semiconsciousness, might have been due to vascular disturbance which led later to intensification of the epilepsy.

Finally, the guinea-pig which had been inoculated with the cerebrospinal fluid died on the fortieth day. Autopsy showed tuberculosis of the inguinal lymph-nodes and of the peritoneum.

SUBACROMIAL BURSITIS—FORMALIN-GLYCERIN INJECTION

Summary: Clinical history of patient; comments; injection of formalin and glycerin; differential diagnosis from chronic synovitis of shoulder; technic of injection; cure by injection; treatment of bursitis by vesicants.

HISTORY

The patient, a male aged thirty-six years, was admitted to hospital January 6, 1915, suffering from soreness at the anterior portion of the right shoulder and from pain when moving the arm.

The present trouble began nine weeks ago, on October 28, 1914. While carrying lumber, soreness developed over the right shoulder. This disappeared, but returned five weeks later, on December 1st, and continues to trouble him. There is tenderness beneath the acromion process, but no swelling. Attempts to place the arm behind the back or to raise it to the head are painful. It also hurts him to lie on the right shoulder. He has had no sore throat and no trouble with the teeth. He does not remember having injured the shoulder. He had no chills or fever with the pain.

COMMENTS AND OPERATION

Dr. Murphy (January 7, 1915): This man has subacromial bursitis. When he brings the arm around to the back, he has the greatest pain: he has no pain in the joint. We shall inject the subacromial bursa with formalin and glycerin. It will not benefit him to inject the joint nor to apply extension. Immobilization would lessen the pain, but it may be cured in a short time by injecting the bursa with a 2 per cent. solution of formalin in glycerin and then keeping the arm quiet seventy-two hours. Then the chemical irritation will have passed away, and with it the inflammatory condition produced by carrying the scaffolding on his shoulder. He has no fixation of the shoulder-joint.

That is a different condition from the patient with chronic synovitis of the shoulder-joint. The synovitis patient complains of pain *in* the shoulder. You give him liniment and the acute symptoms subside, but he returns at the end of six months, ten months, twelve months, saying: "Doctor, I cannot do as much with that shoulder as I did before. It is not painful when I put it through limited motion, but it is extremely painful when I endeavor to get it out of the limited range." Here you have an entirely different condition and a different plan of treatment. Mr. Robert Jones, of Liverpool, has an effective plan of treating an ancient synovitis with fixation, but I would not like to have it tried on my own shoulder on account of the extreme pain. He takes these patients and without an anesthetic places them standing in front of him, puts the patient's forearm on the surgeon's right shoulder; then the surgeon clasps his hands over the patient's shoulder and pulls the patient's shoulder downward while he presses the forearm upward until the arm is straight up alongside the head. You see big, strong men faint when he does this, but at the end of three weeks the patient can do anything with that arm. Our plan is to give the patient ether and then force the arm through the same range of motion. You should make the differential diagnosis and treat the bursitis just as in any position in the body. The injections, as a rule, cure it. You must inject with a small syringe, producing chemical irritation in the entire sac, just as when dealing with tendovaginitis. The injection does not cure it when the bursa is chronically infected.

You see I have gone under the acromion process into the bursal zone. The injection produces a state of chemical inflammatory reaction. He will have great pain the next forty-eight hours, and then the pain will disappear entirely. One injection cures, as a rule. We have reviewed the treatment of the acute stage of subacromial bursitis in the volume on General Surgery of the Practical Medicine Series for 1915, p. 530.

[Note.—The patient left hospital within a week, absolutely free from pain.]

SUBUNGUAL CARCINOMA OF FINGER—AMPUTATION; METASTATIC CARCINOMA OF AXILLA—DISSECTION OF AXILLA

Summary: Clinical history of patient; comments and operation; relation of single trauma to development of carcinoma; the cystic type of carcinoma; baneful effects of traction upon scars; importance of interruption of continuity of constricting scars by interposition of graft of normal skin; operation for cervical tuberculous lymphadenitis obsolete; postoperative treatment of carcinoma; lymph-vessels course over aponeurosis—not through muscle; method of dealing with axilla; exposure of anatomy of axilla; question of removal of all the cancer-cells; use of pectoralis major to prevent pressure upon axilla.

HISTORY

The patient, a male aged fifty-six years, was admitted to hospital January 15, 1915. There is no history of tuberculosis in the family. His mother died of carcinoma of the stomach at seventy-six. He had typhoid fever at twenty.

The present trouble began six years ago, in 1909, with slight serous discharge, reddening, and irritation under the tip of the nail of the right middle finger. There were no chills or fever. A physician removed the nail, but the lesion never healed.

Three years later, in October, 1912, the finger was amputated at the base of the second phalanx. At this time there were no recognized metastases to any of the lymph-nodes of the arm. Two or three days after amputation there was a discharge of pus. The patient was incapacitated five weeks, during which time there were chills, night-sweats, and fever. By the end of five weeks the amputated finger was healed, but he noticed small nodules in the right epitrochlear and axillary zones, which gradually increased in size. In October, 1914, two years after the amputation, the axillary mass was removed. It was the size of a cocoanut. Within three weeks he was well. Six weeks after the second operation he noticed a tumor in the axilla, to the left of the operative incision, which enlarged to the size of a hen's egg; at the same time he noticed a mass at the right elbow. The axillary mass ruptured a month later,—on January 17, 1915,—discharging serum. The next morning the dressings were saturated. He had no chill or fever when it ruptured. He has lost no weight.

Examination of the patient, made on January 18, 1915, by Dr. Murphy showed a fluctuating epitrochlear tumor which resembled cystic metastatic carcinoma. There was also a fluctuating tumor in the axilla, and numerous enlarged lymph-nodes toward the midline, but so encapsulated and so freely movable that they did not suggest metastatic carcinoma. A piece was removed and microscoped, and found to be carcinoma of a rapidly growing type.

COMMENTS AND OPERATION

Dr. Murphy (January 21, 1915): That is the story—long-continued irritation underneath the nail, probably from a "wart." Such irritation is the traumatic etiologic element in carcinoma everywhere except carcinoma of the breast, where one mild trauma starts the carcinoma. Except occasionally in the testis, that is the only place in which a carcinoma occurs from one mild trauma, and never from superlative trauma. One never has sarcoma from superlative trauma, nor from an injury severe enough to cause a fracture. The same is true of tuberculosis: one never has metastatic tuberculosis localized at the site of a severe trauma.

Chronic irritation existed for three years. The finger was amputated, but the epitrochlear and axillary lymph-nodes were not removed at that time notwithstanding that the disease was recognized as carcinoma. See that enormous involvement! There was the first deposit of the metastatic carcinoma. The mass fluctuates distinctly—this is the cystic type of secondary carcinoma. It occurs more commonly in this position than in any other in the body. The carcinoma does not involve the skin zone here as in the mammary zone. In the latter the carcinoma reaches the skin surface in a definite line above the primary deposit, and, according to Handley, it is forced to that position by the accumulation of malignant epithelial cells behind.

I want to keep this new scar separate from the scar in the axilla, for otherwise there will be a long scar in a position exposed to traction, and then the development of a keloid. There is the muscular aponeurosis. The cancer has gone down here in the septum between the two muscles—down onto the main vascular trunks. I am keeping beneath the capsule all the time—you see it is fairly well encapsulated. This mass should be broken down, resembling an abscess, as so many of them do. Above, we have taken out a specimen to be microscoped. You notice the change in that vessel: we want the pathologist to examine it carefully. Is it the result of chronic infection or of carcinomatosis in the vessels? There is a superficial vein, but here the artery pulsates in the field. There is the nerve.

Let the record show that this mass was encapsulated; that there was a large tumor in the primary axillary zone, and considerable edema of the tissues of the axilla; and that I went under the aponeurosis of the muscle, which you see right in the field.

Following the story, what a long period elapsed since the primary lesion, now six years! The primary lesion remained three years in plain view before the finger was amputated, then three years elapsed before radical procedure was instituted. See the artery pulsating! I insert tension stitches to offset the tension there. Traction is responsible for the occurrence of keloid in these scars. All scars occurring across the axillary zone are potentially dangerous. Again, all scars occurring across the popliteal zone are dangerous—dangerous in that they become sensitive; that they develop fibromata, and that they handicap the patient. The longer they are, the more serious they become. You can see that we made a break in the continuity of the large scar on the arm and axilla. If the scar be separated in two or three places and normal skin interposed, so that traction comes transversely to the long axis of the scar, there will be no opportunity for contraction. That is the same variety of scar-change seen in operations for cervical tuberculous lymphadenitis, which operations, fortunately, are obsolete. We have not operated for cervical tuberculous lymphadenitis since eight years, but treat it with tuberculin, but we may incise and curet suppurating individual lymph-nodes.

After amputation of this breast there will be instituted a definite treatment consisting of hypodermic injections of sodium cacodylate in conjunction with high-tension rays. In some cases we give the Coley serum.

It is upon the aponeurosis of the muscle that the lymphatic vessels run, not *in* the muscle. This is not a mammary carcinoma. We remove the pectoral muscles because they are in juxtaposition with the lesion, even though the same tendency to encapsulation is seen up here as down below. We retain the upper fibers of the pectoralis major to fill in the axilla, because it must be thoroughly cleaned out, excepting possibly the vein and artery, which we hope to spare. We clean out the axilla in the less difficult direction, from above downward, including the mass of lymph-nodes at the beginning of the chain. You notice how white the vein appears: it resembles the edema of an infection. There are adhesions all along the vein. We remove the pectoralis minor. Now we are enabled to separate the whole mass from the artery and vein. There is the brachial plexus clearly in view; there, the brachial artery passing between the two heads of the median nerve. We may have to resect the wall of the vein and suture it. There are the anterior thoracic nerves. Now we get into deep water with the vein. We remove the clavipectoral fascia. There is the long subscapular nerve descending. See the size of this mass of lymph-nodes, how it fills the entire space between the muscles! You now see the path by which carcinoma travels—not in, but *between*, the muscles.

Now, you may ask, have all the cancer-cells been removed? By no means. The major portion of the cancer has been taken out, but some of the cancer-cells remain. You recall the splendid paper by Gaylord on that subject. The pectoralis minor is gone, but we have preserved the pectoralis major to fill in this dead axillary space. Many years ago we devised this plan of using the muscle to fill the space, thus protecting the vessels and nerves from pressure.

Let the record show that all the sponges were removed, and that a drainage-tube was inserted.

UNUNITED FRACTURE OF HUMERUS—IMPLANTATION;—MUSCULOSPIRAL PARALYSIS FROM SECTION OF NERVE—NEURORRHAPHY

Summary: Clinical history of patient; comments and operation; management of a divided nerve; exposure of pseudarthrosis bathed in blood-serum, in which attenuated infection may lurk; implantation of the fragments; musculospiral nerve sutured; tenoplasty for wrist-drop; shortening of humerus to approximate divided nerve-ends.

HISTORY

The patient, a male aged twenty-six years, was admitted to hospital January 26, 1915. His family history is negative. He has had the usual diseases of childhood. In 1907 he was operated upon for appendicitis, and the appendix was removed.

The present injury was received seven months ago, in June, 1914. While driving a motor truck the steering apparatus broke and the patient's left arm was caught between spokes of the steering wheel, resulting in a comminuted fracture of the humerus just below the middle (Fig. 174). He was taken to a hospital, where the fracture was set, and, twenty-four hours after the accident, it was wired (Figs. 174 and 175). A splint was applied from shoulder to elbow. Three months later, in September, 1914, owing to non-union, the arm was reoperated upon and a piece of dove-tailed bone, 4½ inches long, removed. This time the humerus was plated (Figs. 176 and 177), and the arm dressed in a gypsum case. Upon removal of the gypsum case there was still non-union, and wrist-drop had developed (Fig. 178).

The patient has now wrist-drop, and is powerless to extend the hand or the fingers. Flexion is good. He can pronate and supinate the hand, and can move the arm at the elbow.

COMMENTS AND OPERATION

Dr. Murphy (January 27, 1915): When did he notice the wrist-drop?

Intern: After the operation at which the fracture was plated, upon removal of the gypsum case.

Dr. Murphy: Yes! That is the all-important fact, and now that there is non-union the wrist-drop is easily remedied. He has a divided musculospiral nerve, and the physician frankly told the patient what had caused it. It was not a sequence of the accident.

If the nerve is divided, and if we can expose the ends and contact them, we shall get splendid restoration of function. With the neurofibrillæ exposed like brush-ends and united in accurate apposition, there is the same chance of securing union and restoration of function as when bone is approximated under similar conditions, so regular is the result. Take, for example, the brachial plexus. We have had two cases of division of the brachial plexus above the clavicle, and even higher, in the main trunks. These nerves were united. One man was a laborer and the other a hotel porter. Both have returned to their former positions. [Clinics, June, 1912, p. 339; April, 1913, p. 259; February, 1915, p. 77.]

Upon examination no neuroma could be determined. With the parts now exposed, nothing but cicatricial tissue is seen in every direction. Now we apply the excitor to see whether the nerve is cut above or below. The superficial fibers seem to be intact: the question is whether that is due to a short-circuit through the muscle. Now the nerve is more freely exposed: there are the fibers that supply the triceps. There is no transmission to the muscles of the wrist at all. If the nerve is not cut, but compressed, there is merely suspension of transmission without degeneration and a positive response is obtained by the faradic current *below* the point of compression. The faradic reaction is negative here. That means the nerve is cut somewhere, but just where we do not know yet. No neuroma is apparent in this field, which is against the nerve being divided in *this* zone. We recall a patient from Milwaukee, many years ago, with wrist-drop,

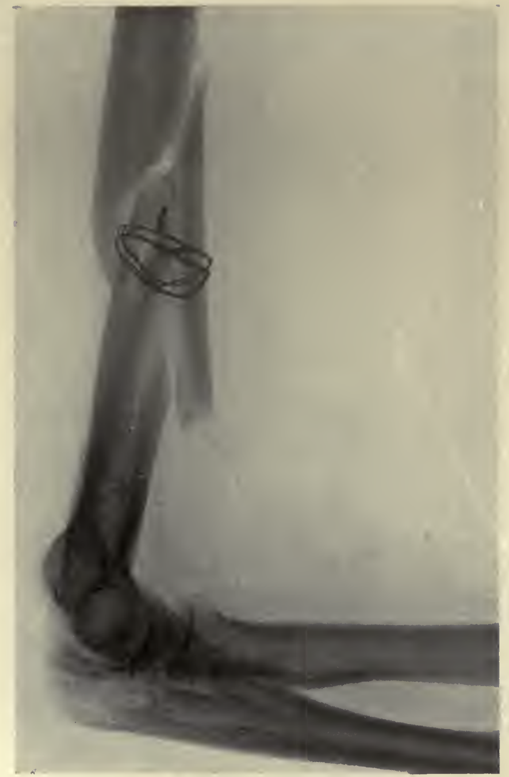

Fig. 174.—Comminuted fracture of the humerus wired twenty-four hours after the accident. Shows dove-tailed fragment that was removed at the second operation (view from outer side).

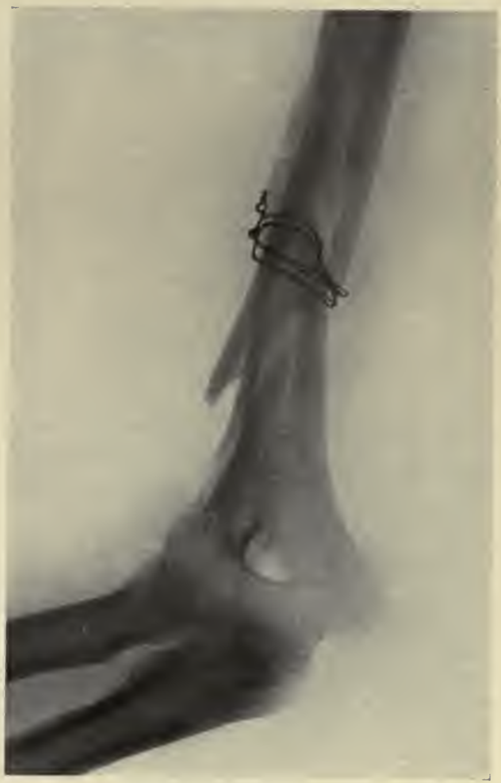

Fig. 175.—Comminuted fracture of the humerus wired twenty-four hours after the accident (view from inner side).

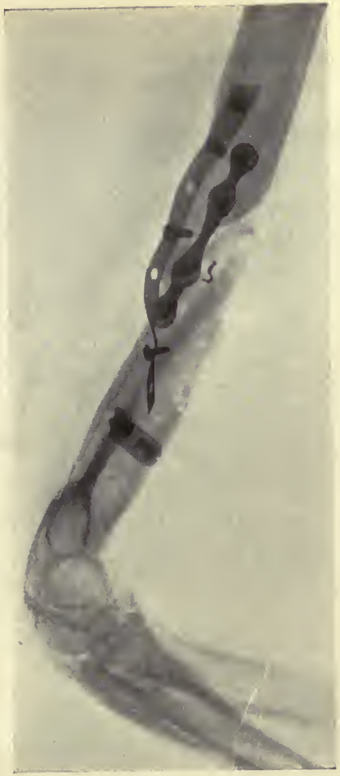

Fig. 176.—Ununited fracture of humerus after second operation, three months later. The dove-tailed fragment shown in Fig. 174 has been removed, and the fragments were plated elsewhere (view from outer side).

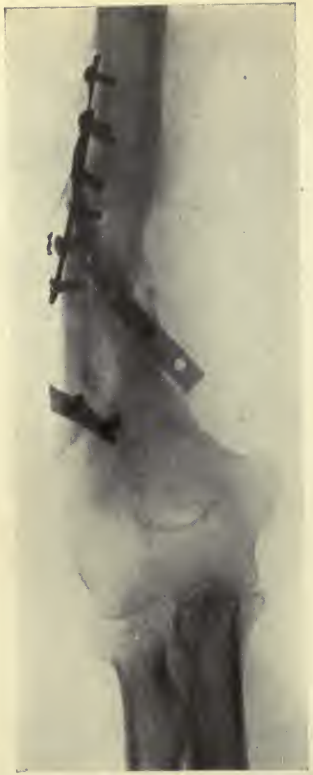

Fig. 177.—Ununited fracture of the humerus after second operation, three months later. Note displacement of plate and metal band from loosening of screws by absorption of bone around them. Note absence of callus (anteroposterior view).

whose musculospiral nerve we exposed, just as we did here, without finding a neuroma. We freed the nerve from the bone callus, and there was immediate faradic response from below the point at which it was fixed, but none from above. That meant it was living and its fibers were compressed only.

Here is the fibrous tissue on the bone, forming a pseudarthrosis. There is the blood-serum one finds around these foreign bodies, sometimes infected, sometimes not. Microörganisms of low virulence are often demonstrable, and when present, create a disturbance. Novy recently recognized them as specific microörganisms. A patient who had heard me speak about that type of infection sued his doctor. I was called as a witness, because the case was published. It was clear from these reports exactly what we meant, but it was not interpreted correctly by the patient. We had occasion then to say that the criticisms in these clinics were criticisms from the standpoint of text. If you held preachers responsible for living up to their text, there would be but few left. The same thing is true of doctors living up to their text in teaching.

Now the bones are fairly well exposed. There is an element of danger in the peculiar fluid that you saw: we feel that we know little or nothing about it microscopically; clinically we are fairly well informed. We work now with the nerve in a wide-open field, lest it be injured. Here is the plate encircling the bone. That is not a real plate: it is merely a metallic strip. We free the periosteum for half an inch above and below the line of fracture. This is a pseudarthrosis. There is the ligament that develops around a pseudarthrosis. This is the condition we imitate in our operation of arthroplasty.

Let the record show that a fragment of muscle is interposed between the ends of the bone.

Now the fragments are exposed, and we press them together to relieve the nerve from tension. There is a fragment of bone that went forward around the nerves and arteries in front. You see how cautious we are, because we do not know what structures are bound together in that zone of adhesions. Now the fragment is free. There is a fragment of bone which will be of service later. What we are figuring on is to expose the nerve throughout its length, and to shorten the humerus in order to free the nerve from tension. The key to success is not to touch it with one's hand. We shall implant this lower fragment into the upper one. These small fragments are left in because of their value as centers of osteogenesis. Now, you see, one fragment is invaginated into the other (Fig. 180). We turn these old screw-holes to good advantage by passing the wire in and out of them. Implantation has many advantages over transplantation as the ends are all living and afford a greater resistance against infection than a transplant would.

Now we must go over this nerve to see if it is divided higher up. There are the fibers of the triceps. Now, since the constrictor has been removed, we must work in the presence of blood, which retards progress. There is the trunk of the nerve again at the uppermost limit of the field. There is no neuroma at this point. There is the branch that supplies the triceps. There the nerve was cut, at the upper part of the incision, where the upper screw was put in: here is a neuroma. Now it shows up beautifully. We cut off the neuromatous ends of the nerve back to the normal fasciculi: see them stick out! We are ready for the approximation. It does not matter whether the proximal axons are contacted with their corresponding distal axons, so long as they send out impulses, since they will regenerate and finally learn to send impulses along the right line. The work here is slow, but were we working for the usefulness of our own arm, the time would not cut much figure.

We sew around the nerve folds of triceps muscle, so it will not be exposed to cicatricial contact. There has been some oozing which has now subsided.

Let the record show that we first exposed the musculospiral nerve at the point of pseudarthrosis and found no neuroma; that we then opened up to the highest point, and just over the last screw-hole found the divided trunk with the neuromata; that we excised a portion of the nerve and contacted the ends with chromicized gut; and that we succeeded in doing the entire work without hand-contacting.

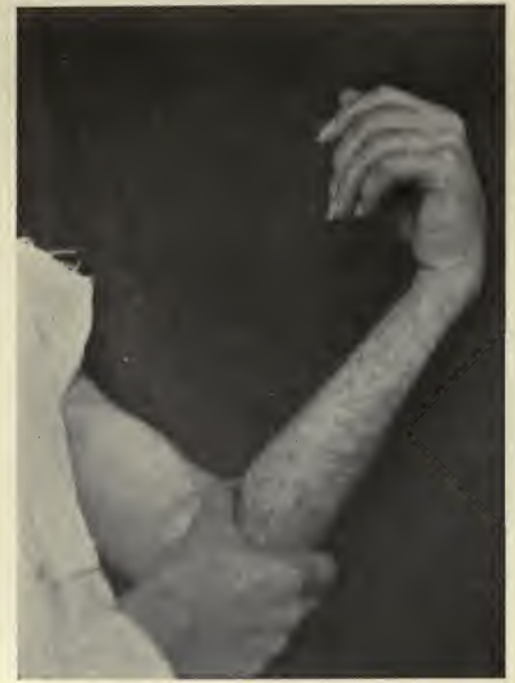

Fig. 178.—Wrist-drop from musculospiral paralysis. Note fingers fixed in flexion. Before operation of neurorrhaphy (view from inner side).

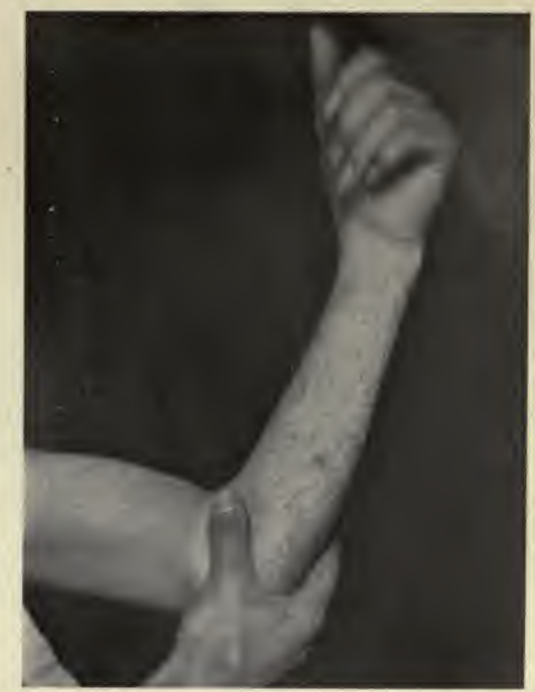

Fig. 179.—Wrist-drop from musculospiral paralysis. As hand straightens by gravity the fingers are completely flexed by the contracted and unopposed flexors. Before operation of neurorrhaphy (view from inner side).

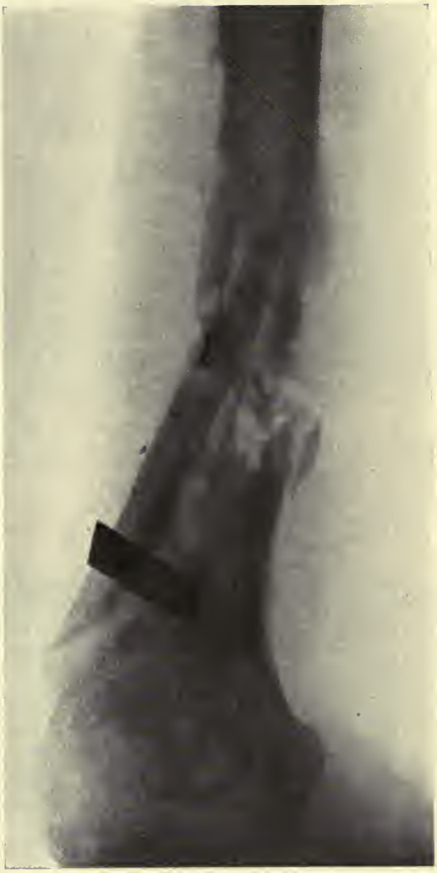

Fig. 180.—Ununited fracture of the humerus after the third operation of implantation of lower fragment into upper. Small fragments retained to act as centers of osteogenesis (anteroposterior view).

Restoration of function requires from twelve to fourteen months. If at the end of that time no evidences of restoration exist, then we shall perform the author's tenoplastic operation of isolating the flexor carpi radialis tendon and stitching it to the extensor tendons of the digits. This procedure has given excellent results in the cure of wrist-drop. [See next case.]

The humerus had to be shortened one-half to three-fourths of an inch in order to contact the nerve-ends. Immobilization must be maintained from twelve to fourteen weeks.

ANCIENT GUNSHOT DIVISION OF MUSCULOSPIRAL NERVE—TENOPLASTY—ADDITIONAL NOTE

[NOTE.—This case was reported with illustrations in the June, 1914, CLINICS, p. 477.]

Summary: Clinical history of case; end-result in Dr. Murphy's tenoplasty of flexor carpi radialis for wrist-drop in cases where the divided ends of the musculospiral nerve cannot be restored.

DR. MURPHY (January 21, 1915): Here is an interesting case. This boy had a fracture of the right humerus with division of the musculospiral nerve. We exposed the nerve below, followed it up, bringing the arm in a flexed position, and then united it. He was operated upon twice, first in October, 1913, and later, for the nerve condition, in February, 1914. He had a typical wrist-drop. How is that for a result?

We told you that story intentionally—it is untrue, as the restoration of the nerve was impossible, but the result looks as though the nerve had been completely restored. We divided the flexor carpi radialis tendon at the annular ligament, transposed it to the dorsum, perforated the extensor tendon of the thumb and each finger with it, and sutured them to it. We could not bring the nerve-ends together, since there was a great gap of one and one-half inches. There is the flexor carpi radialis tendon. The musculospiral nerve is gone. We think that is one of the prettiest and most effective operations we have ever devised. We have now had four of these cases. The flexor carpi radialis tendon transfixes first the extensors pollicis longus and brevis, second, the extensor indicis tendon,—all at an acute angle to its long axis,—and finally it perforates all the remaining tendons of the fingers at right angles to its long axis, so that now it serves as an extensor for all five digits. We figured it all out. One could not believe, from any description, without seeing it, that a patient could get so perfect a result. When the patient tries to extend the little finger alone, he is powerless to do it. If we first straighten the others, then he can extend the little finger. He brings it to a certain position at which accessory muscles, the lumbricals and interossei, come into play and complete the extension.

In this boy's hand we ran the proximal portion of the flexor carpi radialis tendon vertically and the distal obliquely, so that he could extend the thumb and first finger without extending the remaining three fingers. Why? Because they come into play successively at different ranges of tension, since arranged at different angles.

[Compare the next case with this one.]

In every injury of the musculospiral nerve where union of the cut ends is not possible one may resort to this operation. We have had practically the same result in another class of cases, and have performed many tendon transplantations in the lower extremity for paralysis from anterior poliomyelitis.

ANCIENT FRACTURE OF EXTERNAL CONDYLE OF HUMERUS—DIVISION OF MUSCULOSPIRAL NERVE—TENOPLASTY ON HAND

Summary: Clinical history of case; comments; non-union common at compact areas of ends of bones; two methods of dealing with musculospiral paralysis; conditions for which neurorrhaphy is not available; reasons why neurorrhaphy is contraindicated in this case; fracture of condyle ignored—tenoplasty decided upon; steps of tenoplasty; how it saves time for the patient in rapid restoration of function—one month *vs.* one year; operation easy but time-consuming; results of tenoplasty.

HISTORY

The patient, a male aged twenty years, was admitted to hospital April 15, 1915. He is a foreigner and does not speak English, and the following history is the one which the physician who originally saw the case sent with the patient:

Ten months ago, on June 18, 1914, the right arm was caught in a belt wheel, resulting in injury to the right elbow-joint. Skiagram revealed fracture of the external condyle of the humerus and luxation of the radius (Figs. 181 and 182). When admitted to hospital, there was complete musculospiral paralysis. After consultation with a colleague it was decided to reduce the fracture and dress the arm in hyperflexion. Thinking the paralysis might be due to pressure upon the nerve, an operation was considered. Three weeks later, the paralysis continuing, the splint was removed, and the fragments found in good apposition. Passive motion and electric treatments were instituted. He could extend the arm within a greater arc, but electric response of the musculospiral nerve was negative to the faradic current. Galvanic reaction was positive. Three months after the injury, in September, 1914, the external condyle was much more prominent. This was interpreted as due to too early movements, which increased the amount of callus, resulting in fibrous union. Two months ago, on February 26, 1915, he was operated upon with a view to nailing the fracture. Cutting down through the old scar, the fragments were found to be bound to a large, firm callus and nothing further was done. The proximal end of the severed musculospiral nerve was located, but the distal portion was not found. The proximal portion was split, lengthened, and the end was sutured beneath a muscle-sheath attached to the external condyle. The wound healed, and within a few days it was noticed that the patient could slightly extend the index-finger, which movement was impossible before operation.

The patient is brought here for the purpose of restoring the usefulness of the arm.

COMMENTS AND OPERATION

Dr. Murphy (April 17, 1915): You mean by the old scar what? It was not a compound fracture, was it?

Intern: No, sir.

Dr. Murphy: No, it was not a compound fracture. As a sequence of the fracture of the external condyle, he has a wrist-drop, which was present before the operation: it is not a post-operative wrist-drop, but a traumatic. The physician exposed the bone and stated that he found the proximal end of the musculospiral nerve, but that he could not find the distal end. He attached the proximal end to some muscle-fibers, hoping the neurofibrillæ would be guided toward the distal end. He did not disturb the line of non-union.

The only place in the ends of a bone that non-union commonly occurs is at a condyle. Why? Because in the ends of a bone, with this exception, there is always cancellated tissue. One practically never sees non-union at the site of cancellated tissue. In this position, at the neck of the condyle, there is no cancellated tissue. It is compact, and that is why non-union occurs so commonly in that location. We have had half a dozen, perhaps ten, such cases here from time to time. It is at the beginning of the compact tissue. Were it lower, union would occur, but the

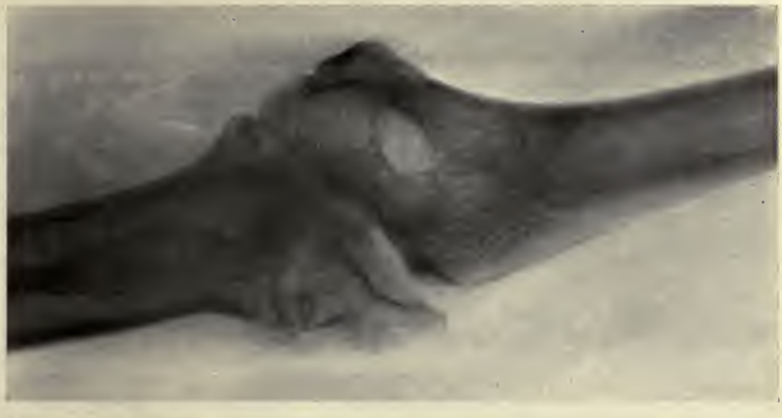

Fig. 181.—Ancient ununited fracture of external condyle of humerus (antero-posterior view).

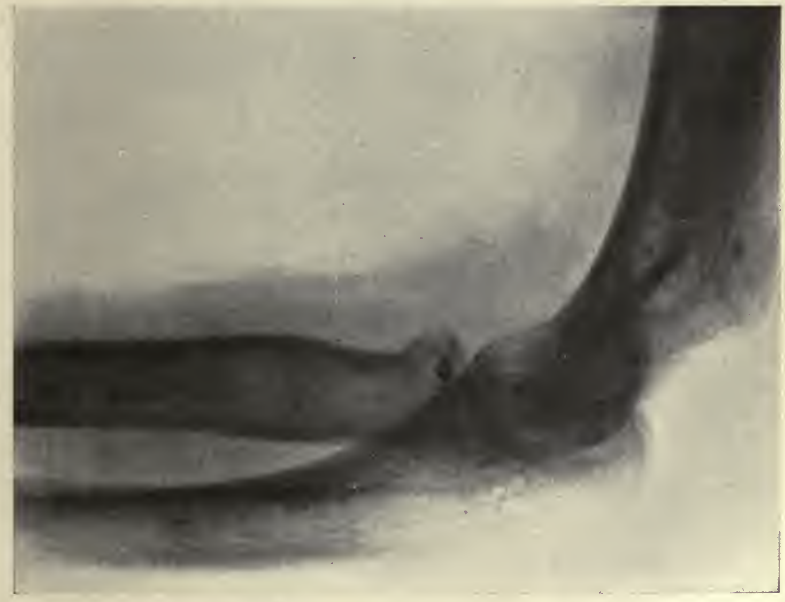

Fig. 182.—Ancient ununited fracture of external condyle of humerus (lateral view).

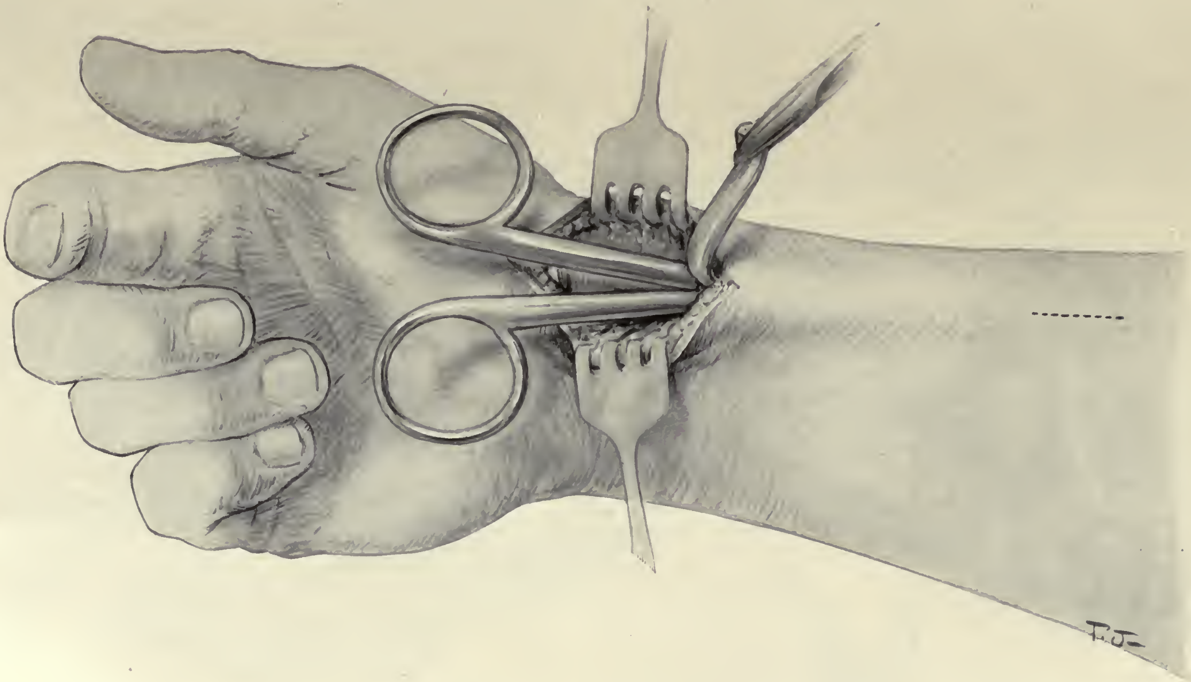

Fig. 183.—Tenoplasty: shows the tendon of the flexor carpi radialis after division just below the annular ligament and elevated. The blunt scissors is passed upward a distance of four inches beneath the skin and on the muscle tendon. It is spread as it is passed. The dotted line indicates the position in which the skin is to be incised and where the end of the tendon is to be drawn out.

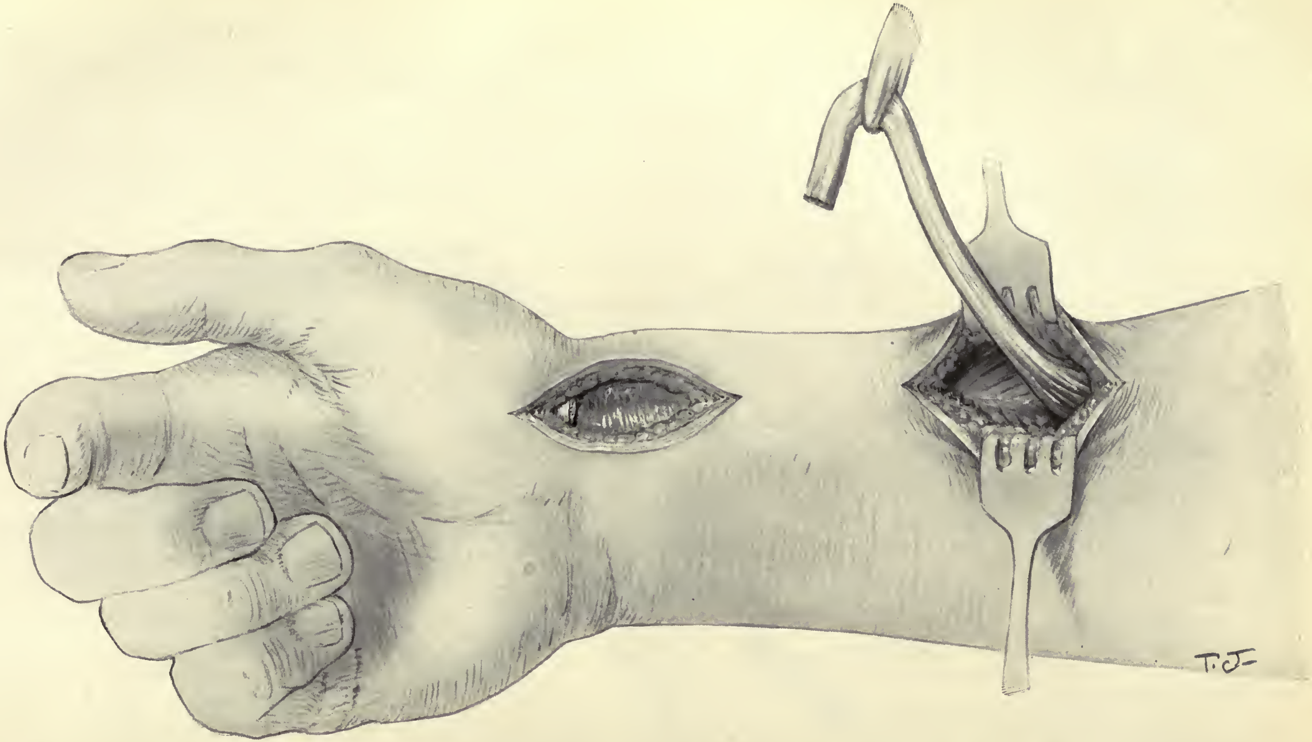

Fig. 184.—Tenoplasty: shows the tendon drawn out in order to pass it over the brachioradialis and beneath the skin, and give it the proper angle for forceful contraction as an extensor muscle.

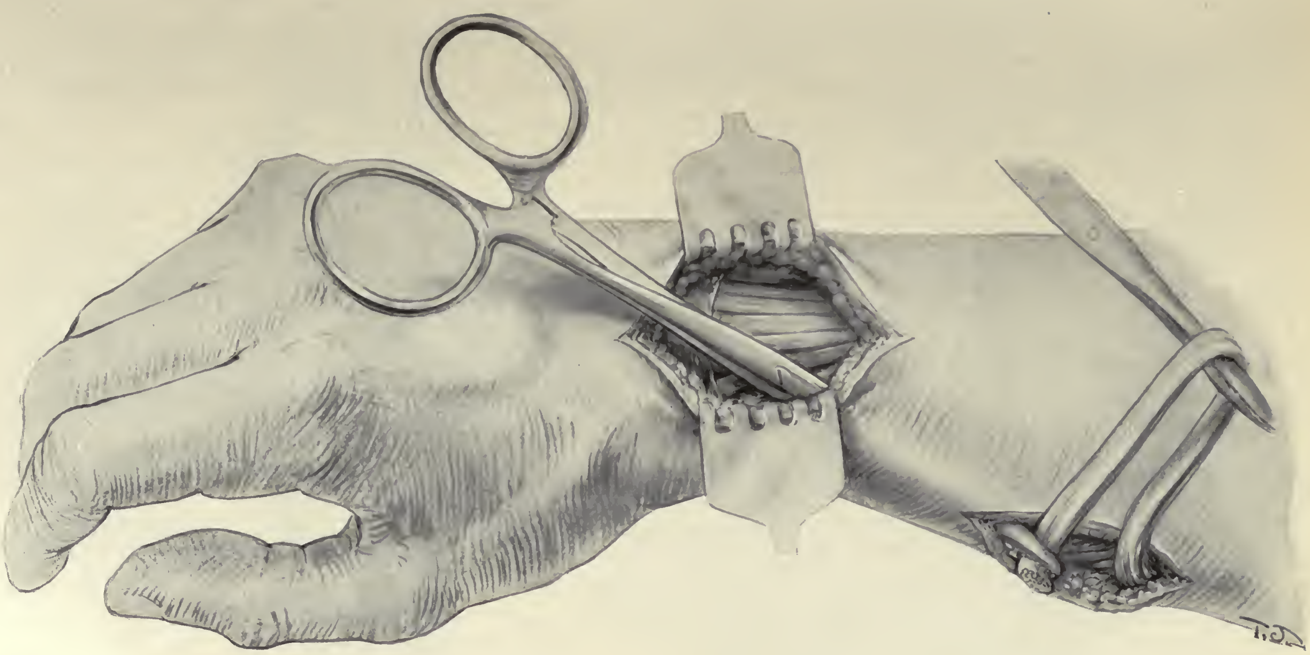

Fig. 185.—Tenoplasty: shows the incision on the dorsum of the hand over the extensor tendons with the forceps passing beneath the fascia and fat, and grasping the end of the flexor carpi radialis tendon.

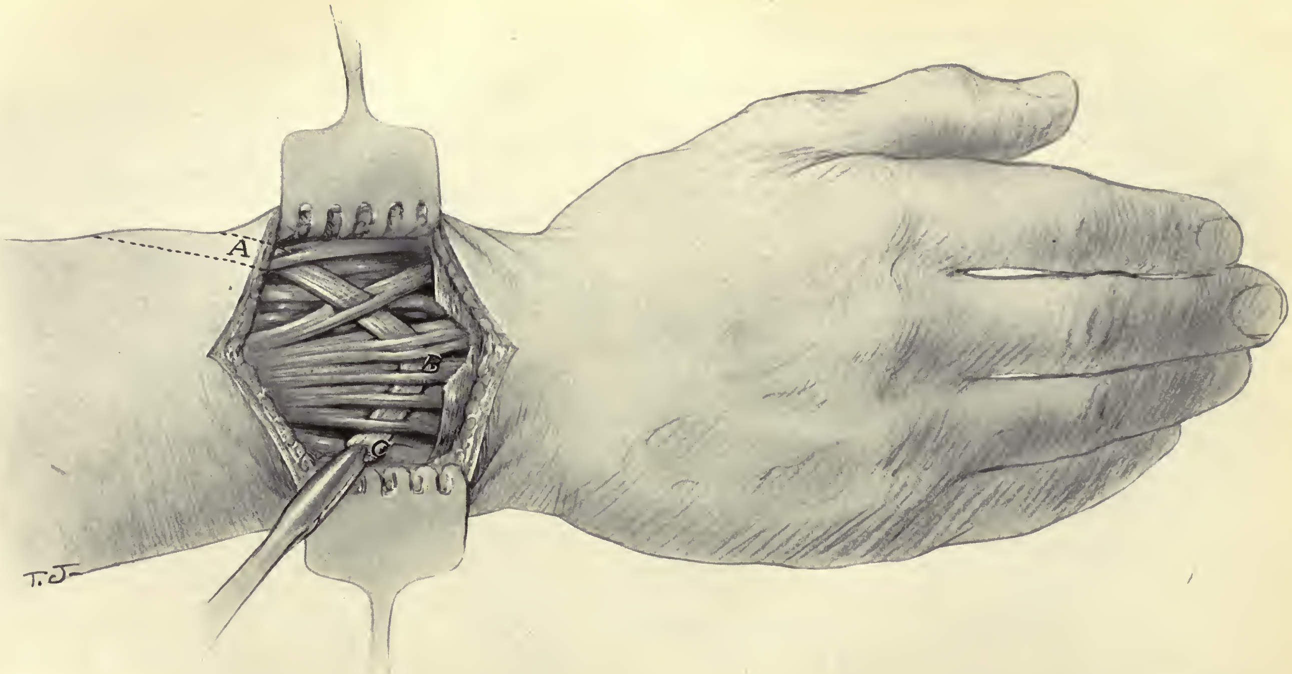

Fig. 186.—Tenoplasty: shows the tendon of the flexor carpi radialis as it passes through the transfixed extensor longus and brevis pollicis, the two extensor tendons of the index-finger, and the extensor tendons of each of the remaining fingers, the line from *A* to *B* being only slightly deviated from the direct traction line of the tendon, so that the primary contraction of the tendon extends the index-finger and the thumb. The tendon from *B* to *C* is at right angles to the portion of the tendon from *A* to *B*, so that the index-finger and thumb may be used continuously extended and contracted without extending the middle, ring, or little fingers. When full contraction and full extension are made, then all five fingers are extended. One cannot extend the little or ring fingers without extending the other three. One can extend and flex the thumb and index-finger without moving the other three.

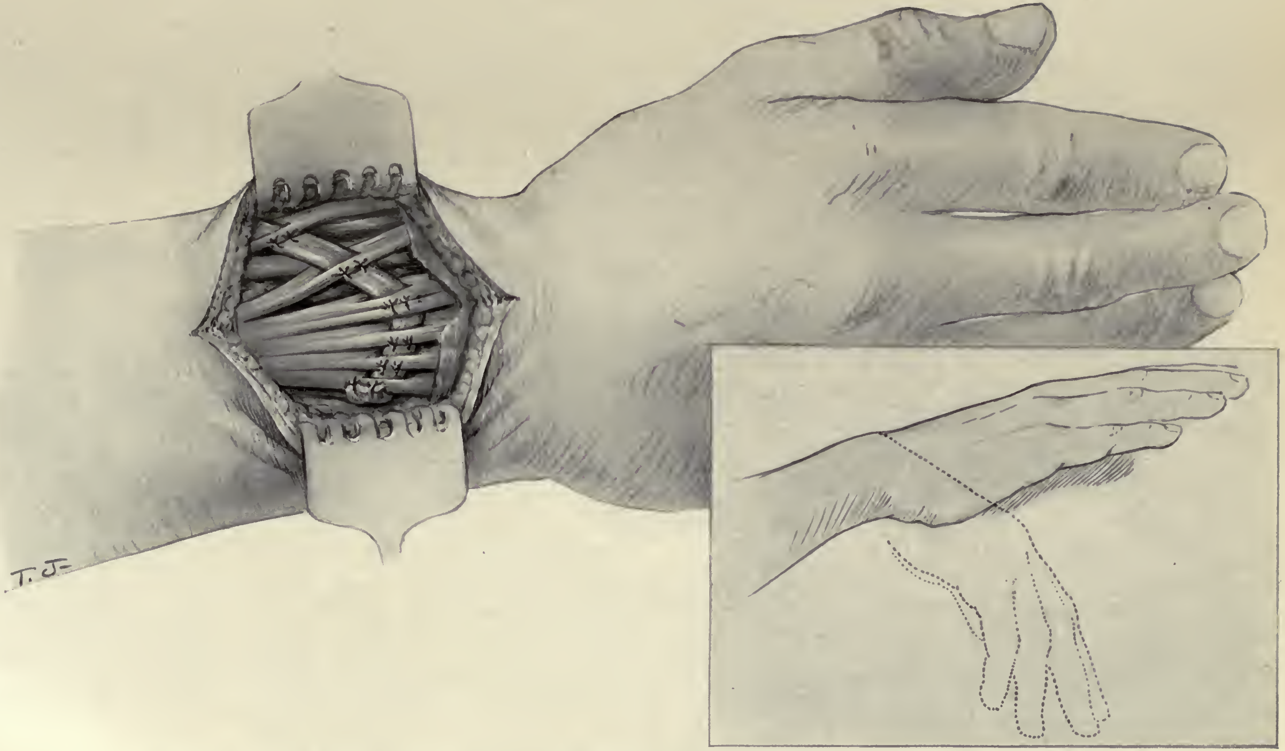

Fig. 187.—Tenoplasty: shows the sutures as placed in the extensor tendons, fixing them to the tendon of the flexor carpi radialis, so that they become an integral part of it. The insert shows the practical effect on the hand at the end of the third day after operation. The patient had full extension of all his fingers and the thumb, and he could extend the index-finger and thumb without extending any of the three other fingers.

line of fracture has involved the densest portion of the compact tissue.

There are two methods of dealing with injury of the musculospiral nerve. The first is exposure of the ends and end-to-end approximation, covering the line of union with muscle, fascia, fat, or egg-membrane, so that regeneration will follow. The conditions under which that is not possible are, first, gunshot wounds of the elbow, which most frequently result in this paralysis. Here the ends of the nerve cannot be found, so that plastic nerve operation is impossible; second, in fracture of the external condyle, which, while it does not always divide the nerve, yet frequently binds it down in a firm callus that blocks conduction; the third is where the nerve is divided completely and retracted from the point of division, which, we apprehend, is the condition here. In the dissection the physician was unable to find the distal end of the nerve. That was seven weeks ago. There are two reasons why we should not try to locate the ends of that nerve. One is, there is a mass of connective tissue to an extent of four inches. We can feel the upper end plainly, but the lower end is buried in a mass of connective tissue. In addition, infection is present, and a sinus leads down to the site of approximation. There is, however, an operation which may be substituted for neurorrhaphy of the musculospiral nerve, and that is tendon transplantation. In a recent case of the latter operation the result was striking. At the International Surgical Congress in April, 1914, we showed this patient ostensibly as an example of reunion of the musculospiral nerve, and every one accepted it as a good result. Then we demonstrated what we had really done. [For plan of tenoplasty see preceding case.]

This operation of tenoplasty is the scheme we are to follow in this case to-day. It is a splendid solution for a very serious condition.

Now we need not interfere with this elbow, for the range of motion is extensive. That he has non-union of the condyle does not concern us at all. He can close the hand, but that is futile if he cannot open it. He has both wrist-drop and finger-drop.

Let the record show that we first exposed the flexor carpi radialis, detached it, and released it dorsally four inches by tunneling through a buttonhole incision; that we passed it subcutaneously downward again to the level of the upper margin of the posterior annular ligament; that we then located and identified the extensor tendons; that, after this exposure, we transfixed the extensor tendons of the thumb and then of each finger—two for the index- and two for the little finger; that the tendons of the thumb and of the index-finger were attached in an almost direct line, and then the remaining tendons were arranged obliquely from above and without downward and inward, so that extension would not bring into play the inner three fingers ahead of the thumb and the index—in other words, so that the thumb and the index would extend independently of the other three; that after completing this step we united the superficial fascia and fat so as to prevent union of the tendons with the skin; and that the skin was closed with catgut sutures and the hand dressed in a superlatively extended position until union occurs.

It can be seen what a time-saver this operation is to the patient. Here we expect complete restoration of function within four weeks, while after nerve approximation twelve months must elapse before function is restored. The results of this operation have been gratifying, and the work is not difficult technically, but just a little tedious from the necessity of minute attention to details.

[APRIL 20, 1915: He can work all the extensors and get the fingers out straight, and it is only three days since the operation (Figs. 188–193).]

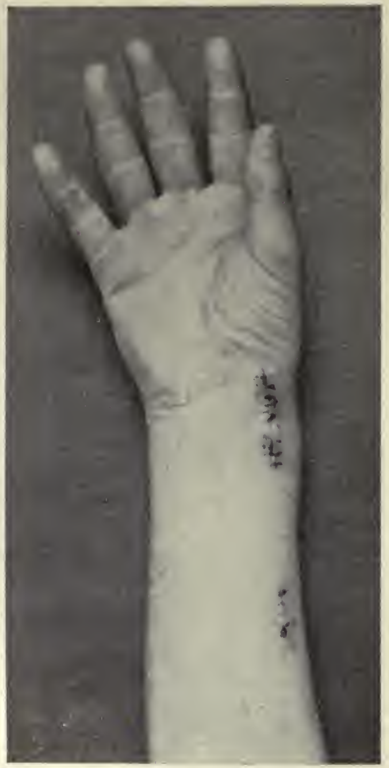

Fig. 188.—Dr. Murphy's tenoplasty for musculospiral paralysis. Note complete extension of fingers and thumb three days after operation.

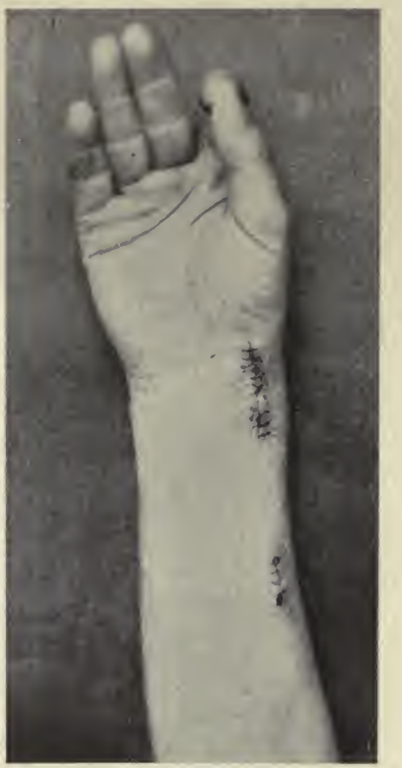

Fig. 189.—Opposition of index-finger with thumb. Note that these two digits extend independently of the remaining three fingers.

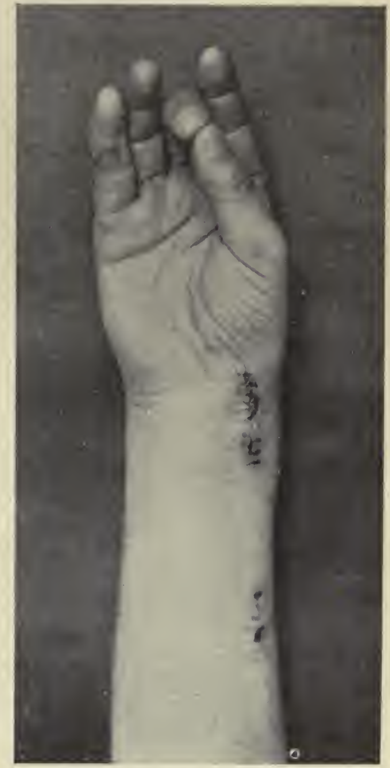

Fig. 190.—Opposition of middle finger. Note independent extension of index-finger from remaining three fingers.

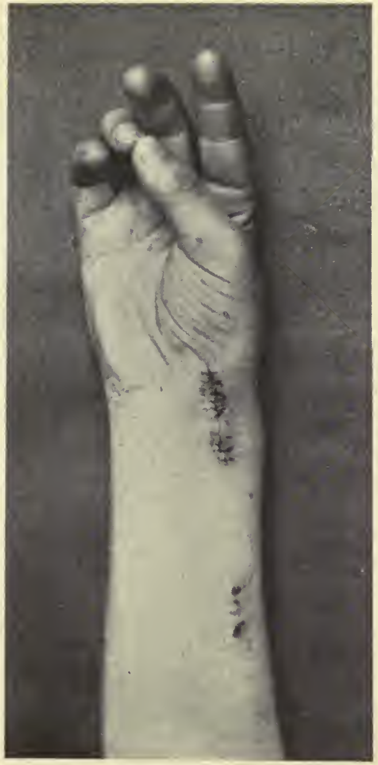

Fig. 191.—Opposition of ring finger.

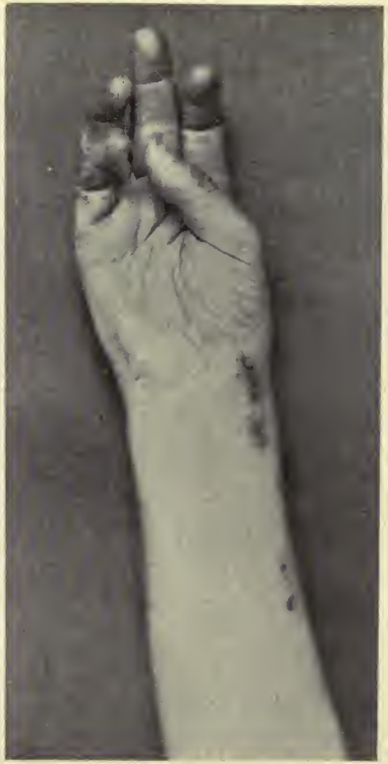

Fig. 192.—Opposition of little finger.

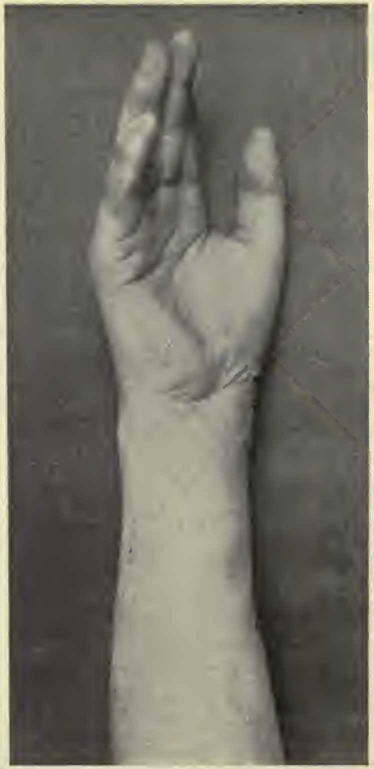

Fig. 193.—Extension of fingers (lateral view).

INFANTILE PALSY OF FLEXORS OF HAND AND FINGERS—TENOPLASTY

Summary: Clinical history of case; comments; paralysis of ulnar and median nerves; steps by which certain tendons are selected for transplantation; adaptation of transplanted tendons; surgical anatomy of tendons, nerves, and vessels about wrist; technic of operation; necessity of lengthening extensor tendons; training the muscles after operation; mode of transmission, difficulty of diagnosis, and necessity of a prophylactic vaccine in anterior poliomyelitis.

HISTORY

The patient, a male aged seven years, was admitted to hospital September 9, 1914. The family history is negative. He has had the usual diseases of childhood.

The present trouble began over three years ago, on July 9, 1911, with severe headache. The next day there was high fever, but there were no chills nor was there any nausea. That night he lay quietly in bed, completely relaxed, complaining slightly of headache. Two days later, while at breakfast, the parents noticed that he was powerless to use the right hand. Sensation, however, was normal. This paralytic state continued, after the onset of the fever, for two weeks, when motion returned to the thumb, and in September, 1911, he could raise the arm over the head. At present he can, in addition, flex and extend the forearm and extend the wrist, but cannot flex the wrist or flex the thumb and fingers unless the wrist be hyperextended upon the forearm.

COMMENTS AND OPERATION

Dr. Murphy (September 10, 1914): He has no power of flexion of the fingers nor of the thumb. What two nerves are out of commission?

Intern: The ulnar and the median.

Dr. Murphy: Yes, they are both out of commission. He cannot adduct the thumb, signifying that the ulnar nerve is entirely out of commission. When he hyperextends the hand, the fingers are automatically flexed through the tendons of the flexor group of muscles. He has power of supination, showing that the brachioradialis is in commission. Extension is intact. If he could close the hand, he would have a serviceable member. What can be done? What muscle can we dispense with as an extensor and appropriate as a flexor? That is the proposition. We have faced this proposition before in exactly the same way. We can take the two radial carpal extensors and convert them into flexors. We do not care so much about the flexion of the hand as the flexion of the fingers. We can take the brachioradialis, which is a weak supinator, and convert it also into a flexor. Therefore, there are three muscles we can convert into flexors, namely, the extensor carpi radialis longus, the brevis, and the brachioradialis. What is the first indication? To secure a good flexor for the thumb. If he could only flex the thumb, the hand would be of more value than it is now. What is the second indication? To provide flexors for the next two fingers, the index and middle. Hence if we can expose and free these three muscles from their insertions and make them play the rôle of the flexors that are out of commission, he will have a serviceable hand. We do not need a new flexor carpi radialis. Why? Because flexion of the fingers flexes the hand secondarily.

The extensors carpi radialis longus and brevis are attached to the bases of the second and third metacarpal bones, after running beneath the extensors of the thumb. In this patient's arm all the muscles are feebler than normal. The next thing is to find the styloid process of the radius. Here is the supinator longus, or brachioradialis, as it is more commonly termed. We shall detach it, bring it forward, and fasten it to the flexor tendons. We now stretch the extensor tendons, so that they will not antagonize too forcibly when we bring the hand forward. He will soon learn to train the muscles in accordance with the plan of the operation, and it is surprising how quickly patients learn this. Dogs adapt their motions in a day after transplantation of their tendons—in a day they learn to contract the proper tendon when an extensor is converted into a flexor.

We find the three tendons here in close proximity. We detach the extensors longus and brevis and let them slip out from beneath the thumb extensors. Here is the tendon of the brachioradialis. Why does it not move the wrist-joint? Because it is attached to the styloid process of the radius. We expose the flexor tendons by going to the ulnar side of the radial artery and nerve. We thus encounter the median nerve. The next step is to isolate the flexors profundus and sublimis. Now we have all the tendons of the superficial group: let us elevate them so as to expose the deep group.

We are ready to transplant. First, however, the extensor tendons, short by contraction from lack of opposition, must be elongated three-quarters of an inch. We have transposed these tendons many times in practically every type of contraction, and so far have obtained good results. We expect to get a good result in this case. The bleeding has stopped. In closing the wound it is important to insert a stitch that will lift the skin away from the spliced tendons, otherwise adhesions are liable to develop between the tendons and the skin.

Visiting Doctor: What is the after-treatment?

Dr. Murphy: Shortly after operation we commence working the hand and training the patient to use it—to extend the fingers and flex them again. He soon learns to do this. Children learn more rapidly than adults, and animals more rapidly than either.

There has been much in the literature about the discovery of the cause and prevention of the infection in these cases of anterior poliomyelitis, of which this is a beautiful example. These researches will have no practical value until a prophylactic vaccine has been produced, because the destruction occurs before the diagnosis is made. It is an infection, which, for some unknown reason, selects for attack isolated cells in the anterior horns of the spinal cord or motor-cell tract. One can see that, as matters now stand, treatment of the infection itself is of no avail. When the child develops headache and fever, one does not know whether scarlet fever, anterior poliomyelitis, measles, gastro-enteric disturbance, or other disease will reveal itself the next day. That it is endemic is certain; that it has some relation to the milk supply is also fairly certain, but whether it is transmitted by the horsefly has not been so definitely settled.

Let the record show that we exposed the extensor carpi radialis longus and brevis and also the brachioradialis; that the brachioradialis appeared imperfectly developed; that we then detached their tendons, and, passing them forward under the skin, brought them out again three inches higher up than where they had passed beneath the extensors of the thumb; that it was necessary to lengthen the extensor tendons three-quarters of an inch and to stretch the posterior joint capsule, in order to admit of complete passive flexion of the wrist and fingers; that we next attached the extensor carpi radialis brevis to the long flexor tendon of the thumb, so as to obtain full thumb flexion; that we then passed the extensor carpi radialis longus through perforations in all tendons of both the deep and the superficial flexors, fixing each one in succession to the longus tendon, and finally that flexion brings into play (aside from the thumb) successively the index, middle, ring, and little fingers.

In our next case of this type we are going to utilize the extensor carpi radialis longus only, appropriating it to the anterior tendons by the method described in the previous case of appropriating the flexor carpi radialis to the posterior tendons. This should be done above the annular ligament, so as not to have extension retarded by the constriction of this ligament on the point of union.

[Note.—There was complete primary union. The patient left the hospital at the end of two weeks.—Ed.]

TUBERCULOSIS OF STERNUM AND RIB—EXCISION OF SINUS AND CURETTAGE

Summary: Clinical history of case; comments and operation; infections of sternum—tuberculous and typhoid; incision and curettage.

HISTORY

The patient, a female aged forty-one years, was admitted to hospital in April, 1915, complaining of a sinus over the lower part of the sternum, which is tender and which aches at times. She had scarlet fever in childhood. There is no history of tuberculosis in the family. Her mother died of carcinoma of the breast. The menstrual history is negative.

Six years ago, in 1909, she was operated upon for cervical tuberculous lymphadenitis. Four years ago, in 1911, a painful lump the size of a hen's egg appeared just below and internal to the left breast. It was opened and discharged pus freely, and an adjacent rib was curetted. The discharge continued for three months until the sinus closed. Her general health was unimpaired. There were no night-sweats, nor was there any cough or loss of weight.

Fifteen months before admission, in December, 1913, a second swelling appeared just to the inner side of the site of the first one. This was incised and curetted, and healed two months later.

The patient remained well until eight days ago, when intense pain from a swelling over the lower portion of the sternum began and lasted four days. The swelling opened of itself and evacuated pus, with relief of pain, but tenderness persists. Since two days there has been aching over the shoulder-blades and in the lower part of the neck, just above the clavicles. During the past year the patient has gained in weight. She still does not cough, nor has she night-sweats. The appetite is good.

COMMENTS AND OPERATION

Dr. Murphy (April 22, 1915): That is the usual history of perichondrial infection, which is commonly tuberculous. The treatment was that routinely employed. A skiagram was taken to demonstrate the sinus.

Of the infections in or near the sternum, the most frequent is tuberculosis, and the most obstinate, that of *Bacillus typhosus*. Typhoid infection of the perichondrium is one of the most annoying things imaginable, and it wears the patient out long before cure is obtained. After repeated operations and after years of annoyance it finally heals. We recall a patient from New Mexico. He remained here two years, and was just one mass of scars on his chest from typhoid infection, but finally recovered. That must have been fifteen years ago, and he has remained well since.

A perichondrial infection of this type may extend from the cartilage into the sternum, later traveling across to the opposite side. It may also attack the sternum posteriorly.

There, you see, is the typical tuberculous granulation tissue, going down into a pocket, where it involves the cartilage.

Let the record show that the sinus was excised and that the carious bone of the sternum and the entire cartilage of the rib were removed with the curet.

METASTATIC THYMUS TUMOR IN BREAST—AMPUTATION

Summary: Clinical history of case; comments; unusual trauma-relationship; cancer of male breast more malignant than that of female breast; characteristic signs and sequelæ of hyperthymusism; amputation of breast; direction of lymphatics from breast; cancer in mice; coffer-damming of cancer-cells; treatment of axilla; the supraclavicular lymph-node; value of x-rays after operation; corpuscles of Hassall in the mammary gland; tumors of the thymus gland.

HISTORY

The patient, a male forty-four years of age, single, was admitted to hospital May 23, 1914. There was no family history of tuberculosis or malignancy. The past history revealed an attack of typhoid fever fifteen years ago—in 1889.

When seventeen years of age the patient frequently struck the right breast with the plow-handle. The breast became sore, especially under the nipple, where a small tumor appeared. A physician painted the breast with iodin and the soreness disappeared, but the lump remained.

Four years ago, in 1910, the breast again became sore, but not from injury. It was again painted with iodin, and again the soreness disappeared, but the lump behind the nipple remained. Since March, 1914, the breast has been painful, and the tumor has increased from the size of a marble to that of a hen's egg, as at present. Chills and fever have at no time been present.

COMMENTS AND OPERATION

Dr. Murphy: This is a tumor in a male breast, which one rarely sees. The history of the ancient trauma, many years previously, is contrary to the usual history of trauma-relationship to neoplasms of the breast. The rule is that within a few years—and often within a few months—of the trauma the enlargement of the breast appears, and often with malignant manifestations.

Here the traumata occurred early in life, and the enlargement was first noticed three years ago. The rapid increase has been during the past year. By palpation one is unable to differentiate between traumatic cavernous angioma with fibrosis and fibroma and malignant neoplasm of the sarcomatous or carcinomatous type. We do not know of any definite statistics on the relative frequency of carcinoma and sarcoma in the male breast.* We know that cancer of the male breast is more malignant than cancer of the female breast, and that a larger percentage dies a cancer death, so that tumors in the male breast demand immediate interference if good results are sought.

There are, however, other features that are not mentioned in the history—the high tenor voice; underdevelopment of the external genitalia, and exceptionally large breasts for a man. There is a considerable quantity of general adipose tissue, but not the great adiposity that is common in thymus disease. The whole picture portrays at once a hyperthymus condition, which increases the hazard of operation, and particularly of the anesthetic. We advised the anesthetist that this man required special attention and careful watching. From this condition of *status lymphaticus hyperthymicus* a certain number of deaths occurs yearly—rarely in adult life, but commonly in infancy and in childhood. He lacks a characteristic that is so common in hyperthymusism, namely, broadening of the hips. He walks perfectly well. Every month or two there are sent to us cases of "hip disease" that are in reality lesions of the thymus gland, and not of the hip primarily. Boys and girls who, at the ages of from twelve to sixteen, weigh from 180 to 230 or 240 pounds, are practically all examples of hyperthymusism. Later, as they approach adolescence, a large percentage develops coxa vara, which, in turn in this instance, is merely a classic manifestation of thymus disease.

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* Dr. J. Leon Herman, of Philadelphia, who is making an extensive investigation into diseases of the breast, kindly supplied the editor with the following figures: Of 1879 cases of mammary carcinoma, 16 occurred in males (1 per cent.).

Of 435 cases of mammary sarcomata collected by Geist and Wilensky (Ann. Surg., July, 1915), 9 occurred in males (2 per cent.).

There is a peculiar resilience in this mammary enlargement that is not suggestive of carcinoma.

[Operation.] We remove the fascia covering the pectoralis major in every tumor case, since the lymphatics course over the surface of the muscle and not within the muscle. The tumor is hard, but not friable, and the gross appearance does not suggest malignancy. We shall not, therefore, remove the lymph-node-bearing areolar tissue of the axilla, nor the pectoral muscles.

The lymphatics from the breast are arranged in four groups, according to the direction they take:

First: The largest group, which passes into the axilla.

Second: The next largest group, which perforates the second intercostal space at the margin of the sternum.

Third: The group that perforates the sixth interspace at the anterior mammary line.

Fourth: The group that travels down to the umbilicus and through the round ligament to the liver. Much clinical importance has been attributed to this last group recently by Handley, whose views we feel, from experience, that we cannot indorse. Therefore, we do not dissect that chain of lymphatics down to the umbilicus, nor do we excise the pectoral muscles.

Most cancer-cells from the breast are carried to the axillary lymph-nodes, where they are arrested and held for a considerable time. Studies of cancer in mice show that metastases travel through the lymphatics and rarely through the blood. It has been estimated that in the Japanese waltzing mouse cancer metastasizes regularly and uniformly on the thirty-ninth day. We do not know how soon after their primary penetration of the basement membrane, which determines malignancy, the cells are conveyed to the next chain of lymphatics. With such knowledge one could say positively when cancer is early and when late. We do know, however, that the time varies with the habit of people. We know that people of corpulent habit do not coffer-dam nor form granulation or fibrous tissue like thin people. They confine the cells in encapsulated, coffer-dammed areas but a short period of time. We know that thin people coffer-dam them many years without showing evidences of metastases.

These observations are not supported by experimental evidence, only by clinical facts. Very early operations have a longer period of permanent immunity and a larger percentage of permanent cures. Fat people and young people have early metastases in distant portions of the body. In them the cancer-cells metastasize rapidly, just as streptococci do. In thin people and old people these cells metastasize like staphylococci. They are held and coffer-dammed in each zone as they progress.

These are the pictures from a clinical standpoint. In the operation, therefore, it is essential that one remove the tumor from the point of primary infection. If the skin be involved in the carcinoma, it should be widely removed. If the skin be free and not fixed to the breast, it is not so essential that a large area be removed. When the growth has penetrated the pectoral fascia and invaded the pectoralis major muscle, the latter should be removed. In all cases, in addition, the loose fatty areolar tissue in front of the posterior wall of the axilla should be removed, because the lymphatic vessels traverse the loose fatty areolar tissue zone. Next, the fatty tissue between the pectoralis major and minor should always be excised, because in this locality there resides commonly one lymph-node, and occasionally two and even three in that little layer of fatty tissue sandwiched between these muscles.

In the radical operation the pectoralis major and minor are detached from the ribs. The pectoralis major is then split from its costal edge directly to the clavicle, or a little below, and thrown outward, leaving it attached at the humeral insertion. The pectoralis minor is likewise detached from the ribs and thrown outward, leaving it attached to the coracoid process of the scapula. Then the fatty tissue surrounding the veins and nerves should be removed up to the clavicle, as well as from the latissimus dorsi and the subscapularis muscles, avoiding injury to the long subscapular nerve which supplies the latissimus dorsi, otherwise in the female the woman cannot move her hand behind and button her belt. The nerve is easily identified. These structures having been inspected and cleaned thoroughly, one should go above the clavicle to remove the supraclavicular lymph-node. According to his statistics, Halsted, who has advocated the most extensive dissection except Handley, has not seen a single permanent cure where the lymph-nodes above the clavicle were enlarged at the operation.

We use the *x*-rays after operation in all these cases, and, if properly administered, secondary recurrences are less liable to occur.

Let the record show that there was a fibrosis of the breast, but no evidence of carcinoma, and that for this reason the axillary lymph-nodes and the pectoral muscles were not removed.

The pathologist reports that this tumor is a fibrous mass interspersed with small cells resembling those of the thymus gland.

DR. MURPHY (May 28, 1914): We have perused the literature bearing upon that case of yesterday, and in the new article written by Klose he cites one case only in which, as in ours, the metastasis from the thymus was situated in the mammary gland. He found the metastasis in the mammary gland not during the course of a special investigation, but as a routine finding in making the section.

In the patient we presented yesterday, in whom there was evidence of thymus disease, we sent the specimen of mammary gland to the pathologist, who reported, knowing naught of the history of the case, that he saw bodies resembling corpuscles of Hassall in the breast. That is a very rare occurrence, for in the most recent work Klose, in *Deutsche Chirurgie*, cites only one case. In the classification on the board it is seen that in myasthenia gravis corpuscles of Hassall occur, but with no symptoms of any other condition and none referable to the thymus gland.

The mammary gland, in which this curious tumor was found in the Frankfort Pathologic Institute, showed the typical cell pattern and the typical cell orientation, so that they felt certain that the diagnosis in that case was correct. This case will be worked out to its ultimate termination. The incidences and conditions associated with malignant disease in the thymus gland have been written upon the board, in addition to the table given above. (See Clinics, October, 1913, vol. ii, No. 5, p. 813.) Here is Rubaschow's collection of 52 cases of sarcoma, which is the most prominent malignant lesion of the thymus gland. Eighteen of these occurred before the age of twenty-five; 8 between twenty-five and forty; and 7 over forty; in the remaining 19 cases no age is given. One of the interesting facts is that in the thymus gland carcinoma and sarcoma occur simultaneously, as reported by Benda and Rubaschow. Cells of both types of tumors are clearly and distinctly contrasted. All cases reported in the literature have terminated fatally, and all gave the history of Basedow's disease. In our case there was nothing except the voice and the deviation from the normal male general physical conformation to suggest a lesion of the thymus.

Of the carcinomata, there are two forms, as reported by Letulle. First, the small epithelial cells without definite alinement; secondly, the medullary form with flat cells of the epithelial type.

The intoxication manifestations of Basedow's disease are the most conspicuous.

[Note.—July 1, 1915. The case progressed without untoward symptoms and has remained well to date.—Ed.]

BRONCHIECTATIC CAVITY—COMPRESSION OF LUNG

Summary: Clinical history of case; comments; lesions that may simulate a bronchiectatic cavity; ability of lung to resist infection; dangers of paracentesis pulmonis; coffer-damming of pleura preparatory to second stage of operation; danger of infecting the pleural cavity; treatment for a pulmonary abscess situated centrally; slow development of surgery of lung; effect of pneumothorax; protective value of a mediastinal septum; nitrogen-gas immobilization of lung.

HISTORY

THE patient, a male, single, aged twenty-five years, was admitted to hospital on September 22, 1914. The family history is negative. There were the usual diseases of childhood. He had tonsillitis twelve years ago, and three attacks of laryngitis within the past ten years.

The present illness began ten years ago, in January, 1904, with an attack of bronchitis. This lasted six months—until July of the same year. He coughed slightly for the first three or four weeks, and as the cough increased he expectorated thick, creamy sputum. Hoarseness accompanied this attack. The temperature was not taken. He did not lose weight. He felt well and strong. The cough gradually ceased, and with it the expectoration.

Four years ago, in 1910, he exposed himself to cold weather after ether anesthesia. Two days later he developed another attack of bronchitis similar to the first. At the end of one year he noticed that the sputum was brick red in color, as though blood-tinged. He immediately submitted a specimen to a pathologist, who reported blood present. Since that time the cough and expectoration have not ceased, nor even moderated. After violent exercise the patient notices more streaks of red blood in the sputum. He does not recall having had fever or night-sweats. He has gained in weight and feels well.

COMMENTS AND OPERATION

Dr. Murphy (September 26, 1914): The analysis of these cases is important. The lesions that may be confused with a bronchiectatic cavity are:

First: Circumscribed interlobar abscess, that is, interlobar pleurisy.

Second: Circumscribed gangrene of the lung. There may be gangrene of the lung with encapsulation of the gangrenous portion and of the abscess, so that there is no expectoration of putrid or offensive material. There may be also recurrent expectorations from the circumscribed gangrenous abscess, that is, it ruptures into the bronchus, empties, and then heals temporarily. Again, it may not close, and the patient continues to expectorate.

Third: There are the circumscribed pleurisies in the periphery of the lung, as well as those that occur in the interlobar zone. All may cause symptoms resembling those of a bronchiectatic cavity.

One of the interesting features in this case is that this expectoration was extremely offensive, suggesting a circumscribed necrotic zone in the lung around the bronchus, which empties back into the bronchus. With empyema or interlobar pleurisy the rule is that the first expectoration is not offensive, but the subsequent expectorations are offensive, and continue to be offensive. Why is it that there may exist in the lung a non-tuberculous abscess so large as this, with expectoration of a pint or a half-pint of pus daily, without impairing the patient's health? It is because the lung has an intrinsic power of resistance. Why is it that the patient does not show more symptoms of intoxication? Aside from the peritoneum, the lung has a greater power of resistance to infection, a greater power of encapsulation and of preventing absorption, than any tissue in the body. The lung resists tuberculosis longer than any tissue in the body except the peritoneum. Why? Have you ever attended the autopsy of a patient who died from pulmonary tuberculosis, and have you ever lifted up the lung and found it as heavy as, if not heavier

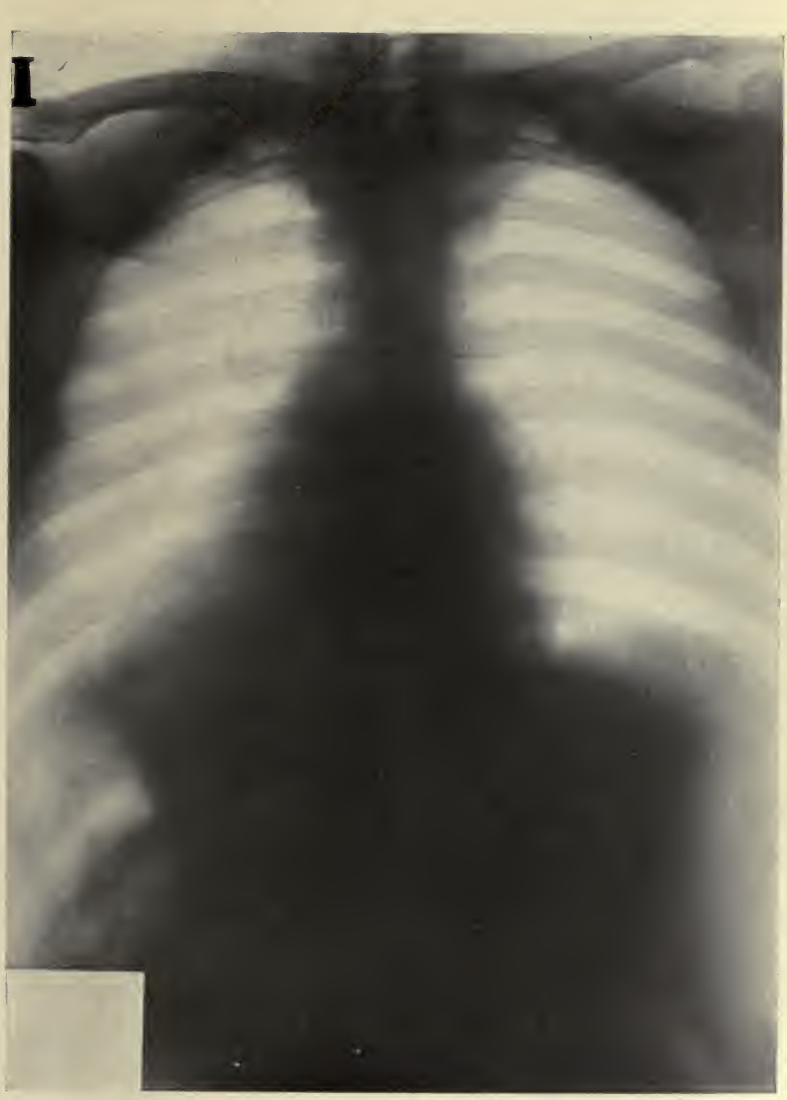

Fig. 194.—Skiagram of thorax showing a bronchiectatic cavity.

than, the liver? What does that excessive weight mean? It means the formation of connective tissue in an endeavor to encapsulate the lesion. It is an expression of resistance. That is why a circumscribed cavity may remain in the lung months and even years with practically no absorption, and with no chills or fever, thus differing entirely from a knee-joint undergoing destruction from tuberculosis with mixed infection. In acute tuberculosis of the lung with mixed infection the process does not encapsulate nor does the disease coffer-dam sufficiently to stop absorption. Therefore we have fever for weeks and months.

We have made no puncture to locate the abscess cavity. Formerly we punctured these lungs for diagnostic purposes. Now we rarely ever puncture, and we have had some painful reasons for not doing it. We recall a case very similar to this which we did puncture with a small exploratory hypodermic needle. On removal of the needle the patient coughed several times and said, "I am dizzy." He soon had a convulsion. What had happened? We had passed the needle through a vein in the lung and then into the cavity. The needle-hole remained patent between the vein and the cavity, and upon coughing he caused air to pass into the vein and had a cerebral air-embolism. He had hemiplegia, from which he died some days later.

We shall open the chest over the small area of flatness to determine whether the lung is adherent to the parietal pleura, and if it is we shall then open directly into the abscess cavity. There is the eighth rib. We enter between the seventh and eighth ribs. The pleura is not adherent, for the lung is seen moving to and fro.

Let the record show that there is a sense of induration in the lung to the inner side of the opening in the thorax, and that the pleura is not adherent.

We shall just pack that space now and do nothing further for five days, by which time he will have developed a reaction in the pleura that will coffer-dam it and lessen the danger of infection. We insert one strip of nosophen gauze.

Remember that acute infection of the pleura, as from opening an abscess, in the absence of infiltration or adhesions in the pleura, is a dangerous sequel, just as in the peritoneum. From the interpretation of the *x*-ray, we expected to find the pleura adherent, but now see that it is not adherent. We shall, therefore, close the opening in the chest by fixing the muscles back into position with heavy silkworm-gut sutures. We shall treat this case by nitrogen compression and favor drainage through the opening in the bronchus.

MONDAY, SEPTEMBER 28, 1914: If the abscess is situated centrally in a position inaccessible for draining or emptying, we shall simply induce pneumothorax, and maintain it by administering nitrogen through the needle. If the abscess can be reached, we shall drain it externally. We cut another rib, so as to provide plenty of space. We have taken out one rib—the next rib we merely cut. If necessary, we shall take out the other rib so as to pass an entire hand into the chest.

The surgery of the lung has developed slowly, because the pulmonary disease we are most often called upon to treat is tuberculosis, and surgery does not control that lesion. The drawing of the lung outside of the chest was accomplished four hundred years ago by a bull in Bologna. He did it by attacking a man, tearing his chest open, and pulling the lung out upon the surface. The good and wise physician who treated him did not try to reduce the lung into the chest. It plugged the opening, contracted into a cicatricial mass, and just stayed on the surface. From that time to the present but few lobes of the lung have been removed, notwithstanding that crude demonstration of its feasibility.

Pneumothorax was the first thing to be feared, but after we demonstrated in 1896 that it was easily controlled in a large percentage of the cases, it was not dreaded so much. In 1896, 1897, and 1898 we showed upon dogs that the chest could be opened by immobilizing the mediastinum, and that the dogs suffered no ill effects. We have such an enormous respiratory reserve that the respiratory exchange in one lung, as is demonstrated by a pulmonary effusion large enough to displace the mediastinum over to the opposite side, may be diminished or exhausted, and still there remains ample respiratory exchange. It has been demonstrated theoretically that a patient at rest has 223 cubic inches of respiratory exchange, while a person in activity has as high as 3600 cubic inches. This shows the enormous range and the enormous reserve that exist in the lung. If one lung only be put out of commission, there is still ample respiratory exchange, but if both lungs be put out of commission, danger arises. If the pleura be opened on both sides and the lung allowed to contract, the patient dies, unless oxygen flow into the bifurcation of the trachea. In our experimental work we found that dogs had a very thin mediastinal septum. If one compress the septum for five, ten, or fifteen minutes, it finally ruptures, air rushes into the opposite side, respiratory exchange is suspended, and the dog dies. None of the monunguates has a mediastinal septum. The horse has no mediastinal septum, and if gored so the air may pass freely into the lung, always dies, because both lungs collapse. It is different with the cow. If a cow be gored, only one lung collapses, because there is a mediastinal septum, and the cow lives on.

We cannot drain this cavity transpleurally without protecting the pleural cavity. We have waited for an inflammatory reaction to take place in the pleura and form adhesions to the chest-wall, but it did not develop. We are therefore now not justified in opening a dilated bronchus—an infected bronchiectatic cavity—letting the pus enter and contaminate the pleura that is free from adhesions. Remember, pleura is more dangerous when infected than peritoneum, because it has no resistance. Of the three great serous tunics, there is the highest type of resistance in the peritoneum and the least type in the meninges, while the pleura occupies the intermediate position.

We remove the packings of the previous operation and sponge the cavity with a 5 per cent. phenol solution.

Let the record show that there were no adhesions of the chest-wall in any position, and that the lung is fully collapsible.

Plugging an opening that one has made in the chest-wall stops the ingress and egress of air, so that the mediastinum becomes immobilized, and the symptoms of dyspnea subside at once. In this case there is now too much air in the pleural cavity and we shall aspirate it. You heard us give the order to have the aspirator ready before starting the operation, so that we could meet this emergency.

If you have read over the experimental work we mentioned, and if you are familiar with the lung, you will feel perfectly at home in chest operations. That gives you assurance.

Let the record show that we opened the chest cavity; that there was a pint of effusion as a sequence of what we did a few days ago, which emphasizes how quickly this transudate takes place; that we then opened the pleura and found no adhesions; that a scar on the lung indicates clearly the site of the bronchiectatic cavity (Fig. 178); that as the abscess is encapsulated and no adhesions exist, it may be more safely treated by pneumothorax and thus drained through the bronchus; and that drainage will be maintained by injections of nitrogen into the pleural cavity. Let the patient lie constantly on the affected side.

TRAUMATIC CERVICAL SPONDYLITIS—CERVICAL NEURITIS—ALCOHOLIC INJECTION OF GREAT OCCIPITAL NERVES

Summary: Clinical history of case; comments; traumatic spondylitis—frequency of involvement of various zones; mistaken diagnoses of the radiating pain; sciatica a symptom, not a lesion; blocking of great occipital nerves; motor nerves must not be injected with osmic acid or alcohol.

HISTORY

The patient, a female aged thirty-five years, was admitted to hospital November 2, 1914.

Her mother and sister died of pulmonary tuberculosis. Menses began at fifteen years, are regular, of the twenty-eight-day type, last four days, and are not painful. The latest period was on October 18, 1914. Four years ago she had mumps which lasted nine days. She has been deaf for years. She is myopic in the right eye and hypermetropic in the left.

Four years ago, in 1910, she was struck upon the head by the cylinder of an engine, which felled her to the ground. She was not stricken unconscious, but got up and walked. A half-hour after the accident four stitches were inserted into the scalp over the right eye. One month later vision in the left eye deteriorated. She noticed dull occipital headache and pain in the neck, aggravated by jarring and by motion of the left arm. The pain radiates along the inside of the arm to the elbow. Since six weeks these pains have been more acute and shooting. Three months ago, in August, 1914, there developed in the throat burning, hoarseness, and dysphagia. There is no evidence of a tumor. She has had no convulsions, but is slow in responding.

COMMENTS AND OPERATION

Dr. Murphy: Where is the pain situated?

Intern: In the back of the head and neck.

Dr. Murphy: Yes. It looks as though she had an injury to the roots of the first, second, and third cervical nerves. Here is the best skiagram of the odontoid process we have ever seen (Fig. 195). There is no deviation. There is also spondylitis in the upper cervical vertebræ, which compresses and irritates the spinal roots (Fig. 196). We shall try to relieve the pain by blocking the upper cervical nerves.

This case is instructive in that she has had no brain lesion since the injury nor any mental aberration.

These traumatic spondylitides occur along the entire length of the spine. They are most common in the lumbar zone, next in the lower cervical (neuritis of brachial plexus), then in the upper cervical, and least common in the thoracic zone. In our community spondylitis is most common in the thoracico-lumbar zone—from the eleventh thoracic to the second lumbar segment. In mining districts it is most frequent in the lumbosacral joint, causing that change in the spine which puts the patients practically out of commission. When spondylitis becomes severe, the pain radiates from the roots along the nerves to the front. A considerable percentage of these sufferers has been operated upon, for, in order of frequency, chronic appendicitis, gall-bladder disease, duodenal or gastric ulcer, and some for suspected disease of the urinary bladder. We mention this because it is so common in this community in the fall of the year. It is such a uniform practice to slight the spine in examination. Most examinations are confined to the ventral surface of the body, and the examiner overlooks the fact that the patient has a spine, or that the spine may be the site of the lesion. Many patients have pain radiating down the sciatic nerve, and come here with that diagnosis, having been treated solely for sciatica. Remember, sciatica is but a symptom, and not a lesion. It is a symptom of many lesions. It must be investigated etiologically, not treated as the pathologic entity.

Dr. Murphy (To patient): Where is the most acute pain?

Patient: On the right side of my neck.

Dr. Murphy: She has most pain on the right side, running up around the hair. Note the slowness of speech and the way

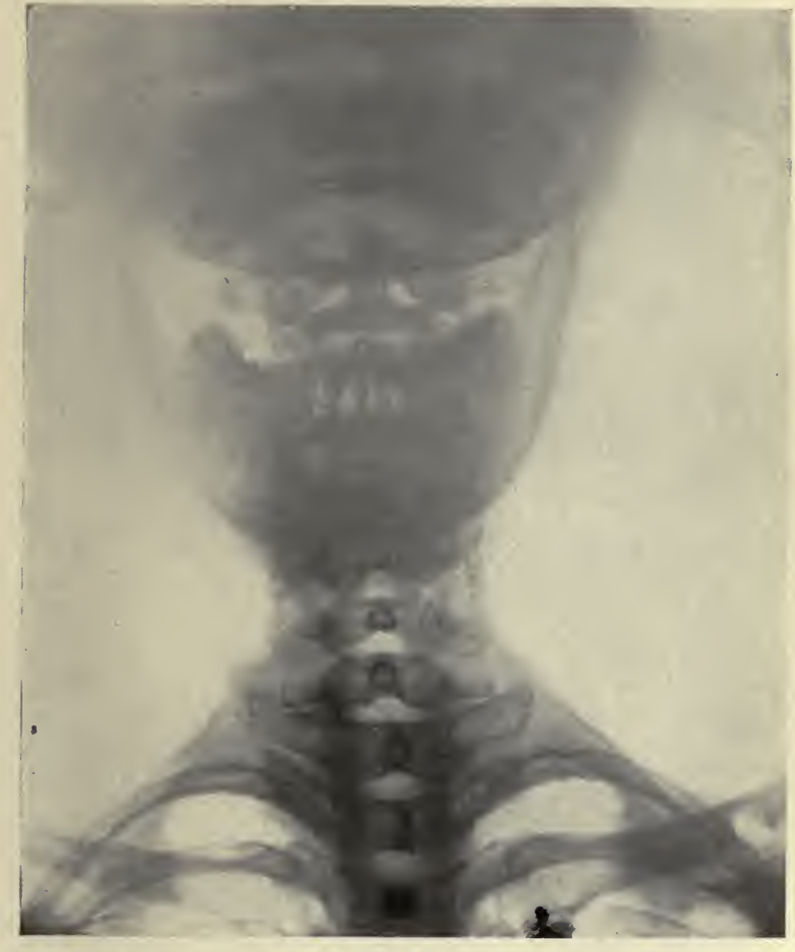

Fig. 195.—Anteroposterior view of skull and cervical spine. The odontoid process, seen through the mouth, is exceptionally well defined.

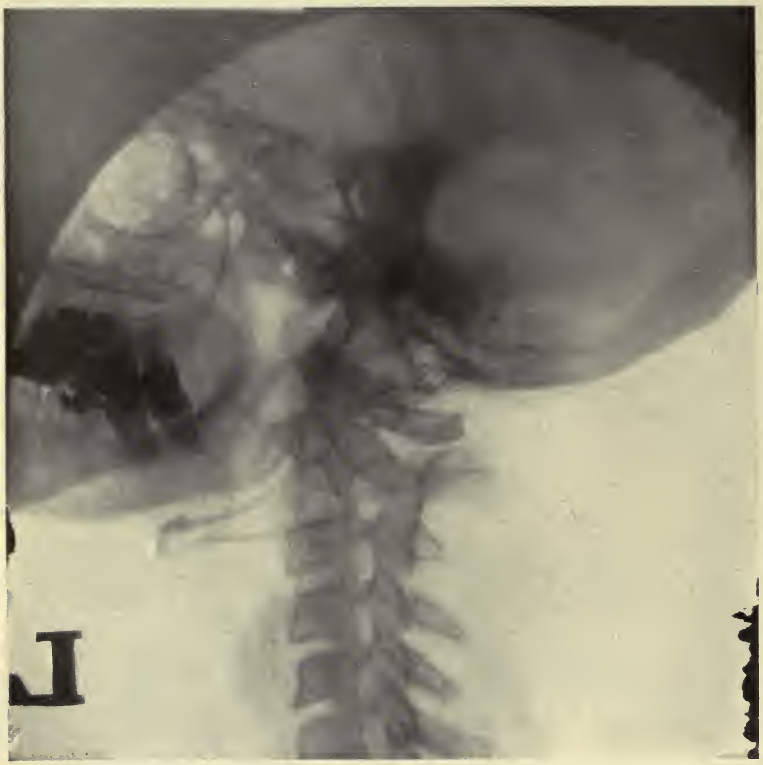

Fig. 196.—Lateral view of preceding figure. There is spondylitis in the upper members of the cervical vertebræ.

in which she articulates. Show me just where you have most of the pain.

[Patient indicates right side of skull, behind.]

Dr. Murphy: That is over the course of the great occipital nerve. One can almost invariably locate it by gentle pressure with the tip of the index-finger. We plan to block it as it comes out from the second cervical nerve. It often receives a branch from the third. That is the spot where you have most of your pain?

Patient: I have pain on the other side, also.

Dr. Murphy: We shall come to the other side. [Inserts needle and injects.]

Patient: That pain shot up into my head!

Dr. Murphy: That indicates that we had the needle just in the right place. This is 70 per cent. alcohol we are injecting. It will be just a little painful for a few moments, and then the pain vanishes. Now we inject the left great occipital nerve. Remember that these nerves are not of importance as motor nerves, otherwise we would not have injected them with osmic acid or alcohol. We receive many letters about injecting osmic acid into the sciatic nerve. The sciatic is a nerve of motion, and must never be injected with osmic acid, because osmic acid and occasionally even alcohol cause permanent paralysis from degeneration of the axons.

Let the record show that we injected 1½ c.c. of a 70 per cent. alcohol solution into the great occipital branch of each second cervical nerve.

[Note.—There was immediate relief of the symptoms and the patient left the hospital free from pain.—Ed.]

BONY TUMOR OF THE SPINAL CANAL—LAMINECTOMY—SPINAL DECOMPRESSION

Summary: Clinical history of case; discussion by Dr. Mix; erroneous conception of effects of suspension of spine upon cord; relief by decompression; analysis of symptoms; result of physical examination; diagnosis between diffuse and transverse myelitis; epicritic and protopathic sensibilities of Head; painful spots of Valleix in intercostal neuralgia; diagnosis of cause of pressure; varying degrees of pressure in *meningitis serosa circumscripta;* final localization of pressure upon cord; comments by Dr. Murphy during operation.

HISTORY

THE patient, a man aged sixty-seven years, was admitted to hospital in June, 1914. His family history is negative for neoplasms. One sister died of tuberculosis two years ago. The patient's past history is negative. He denies venereal infections.

The present illness began two years ago, in 1912. The first symptom was a sharp, shooting pain in the head when putting on his hat. There was also severe, non-radiating pain in the midline of the back, between the scapulæ. After this condition had continued six weeks, gradually becoming worse, the patient consulted a physician, in June, 1913, who diagnosed tuberculosis of the spine. A jacket with a support attached for the head was applied so as to hold the chin up, and was worn for six months, until November, 1913, without benefit. A month later, in December, 1913, he had two chills and some fever, but does not know how much fever nor how long it lasted. He was in bed throughout December, 1913. He never vomited during this attack. After the passing of the attack, in January, 1914, he was weak, but got up and walked about for a few days, eventually resorting to a cane, then crutches, until finally he could not walk. These changes extended over a period of three weeks.

By February 10, 1914, he not only could no longer walk, but the legs were becoming paralyzed. He states that the big toes seemed to carry heavy weights. At this time there was pain in the abdomen, at the level of the umbilicus. The patient said the pain felt like a constricting rope that some one was twisting. When the legs were moved passively, clonic spasms occurred, causing shooting pains in the limbs. These spasms, as well as the abdominal pain, are present now. About three weeks ago urinary symptoms appeared. Since then he has been at times aware of the desire to urinate; at other times unaware. The urine dribbles from incontinence of retention; the feces, also, have been passed involuntarily during the past three weeks; sometimes he is aware, and at other times unaware, of the desire to defecate. These symptoms have progressed in the last fortnight.

The knee-jerks are exaggerated. There was spastic gait so long as the patient was able to walk, and this spasticity was aggravated by the pains in the legs. The pupils of both eyes react promptly to light and to accommodation. The patient has sensation in both legs, but cannot locate the areas of contact, although he knows when they are touched (loss of epicritic sensation and preservation of protopathic sensation). He has never had tenderness over the spine nor signs of spinal deformity.

COMMENTS AND OPERATION

Dr. Murphy (June 16, 1914): Dr. Mix has gone over this case and he will take it up and analyze it with you while we are at work.

Dr. Mix: The patient's trouble began with a series of pains of varying intensity between the shoulder-blades, of the type of root pains, due to involvement of the posterior roots at their entrance into the spinal cord. These pains occurred opposite the spine of the scapula, at the level of the fourth thoracic vertebra, which overlies the fifth segment of the thoracic cord. Beginning in June, 1912, the pains continued and were severe. Soon they disappeared, but subsequently returned. Their recurrence is perhaps an important step in the diagnosis. Disappearance of the pain spontaneously for a time, followed by subsequent reappearance, is frequently due to varying amounts of inflammatory swelling and root compression. When the pains returned a second time they were more severe, indicating increased pressure upon the posterior roots.

The patient then consulted a physician, who requested some skiagrams, which showed the edge of certain vertebræ "eaten away," as he expressed it. It was upon these plates that the diagnosis of caries of the vertebræ was based, and the patient put in a plaster-of-Paris jacket. It was thought that, by relieving the spinal cord of pressure by extension, its function would be restored.

This conclusion was based upon a false conception of the mechanics of suspension of the spinal cord. When patients are suspended by the head to lengthen the vertebræ, the hope is usually expressed that, by straightening the spinal column, and with it the spinal cord, the nerve-roots will be relieved of all pressure, and that, therefore, the symptoms will disappear. This conception is erroneous. The pressure is not due to angulation of the spinal column, but, instead, to the growth of an infectious granuloma, usually a tuberculoma, upon the spinal cord. This infectious granuloma usually originates as a tuberculoma of the body of the vertebra, and often comes through the posterior or posterolateral surface of the vertebra and presses backward. It is the tumor which is compressing the cord, not the angulation of the spine. No amount of suspension nor of immobilization by plaster-of-Paris jackets exerts the slightest effect upon the pressure the tumor bears upon the spinal cord. The only way in which a cord so involved can be helped is by removing the pressure exerted by the tuberculoma. The latter cannot be removed without endangering the life of the individual by scattering the tuberculosis throughout the system. The backward pressure of the tuberculoma compressing the cord against the lamina of the vertebra can be relieved by removal of the lamina. This constitutes a spinal decompression operation, and is exactly comparable to decompression of the skull for tumor of the brain.

The next diagnostic point is that early in January, 1914, there began disturbances in walking. We note the interesting fact that, with exacerbation of the pain, there was exaggeration of the motor symptoms—that the spasticity ebbed and flowed with the pain. In other words, the patient has shown a varying degree of pressure upon the spinal cord, with varying degrees of root irritation and paresis. This was shown on the sensory side by varying degrees of pain, and on the motor side by varying degrces of motor weakness and spasticity.

By the tenth of February the previously intermittent pressure had become so constant as to lead to total paralysis of the lower extremities. At that time, however, there was insufficient pressure to create disturbances of the bladder. Bladder difficulties did not begin until three weeks ago, and they have progressed since then. The patient has incontinence: Yesterday morning, from the time he awoke to the time we saw him, he had three involuntary micturitions. He has no sensation when the urine passes through the urethra, but he does sense the passage of feces or gas through the rectum.

On physical examination we find paraplegia and also paresis of the lower portion of the abdomen. The paraplegia is of the spastic type, and the patient has a positive Babinski reflex in both feet. Ankle-clonus is now present, now absent, apparently depending upon the varying irritation of the spinal cord. The paraplegia and the paresis of the lower portion of the abdominal muscles, the spasticity and the double Babinski reflex, and the occasional presence of ankle-clonus, point to disturbance of the pyramidal tracts. The pressure, however, is unequal because yesterday morning he had a positive Babinski reflex on the left side only. This morning it is present on both sides, indicating that yesterday there was a little less pressure upon the right side than exists to-day, when the pressure upon both sides appears equal.

So far, as we have mentioned, it is evident that somewhere in the spinal cord there is interruption of the conductivity of the upper motor neurons. The question arises whether this interruption extends throughout the cord as a diffuse myelitis or whether it involves one segment alone, as in pressure transverse myelitis. How may we decide between diffuse myelitis and limited transverse myelitis from pressure? Solely by examining carefully the levels at which sensation is disturbed. The sensory disturbances in this patient are peculiar. The intern has noted them. When the intern attempted to find out the limits of sensation he failed, because the patient felt the contact of the pin over both legs. *How did he feel in his legs?* The intern states that he has sensation in the legs, but that he cannot tell what part of them is being touched.

Here, again, we must refer to the well-known observations of Head regarding epicritic and protopathic sensibilities. Protopathic sensibility to touch is primary tactile sensation—mere perception of touch. Epicritic sensibility to touch is something more than mere primary tactile sensation: it is an apperception rather than a perception. Memory and judgment enter into its composition. Now, in transverse lesions the epicritic faculty is lost at a higher level than the protopathic; *and the epicritic loss of tactile sensation usually marks the level of the lesion with accuracy.* In testing protopathic sensation you say to your patient, "Do you feel?" In testing epicritic sensation you say, "I know you feel this, but does it feel natural?"

The patient's epicritic loss corresponds exactly to the level of the third segment of the thoracic cord, which locates it at the level of the second thoracic vertebra. In reaching a conclusion as to the upper level of sensation, we find ourselves unable to demonstrate the level of epicritic loss along the spinal ends of the nerve-trunks, but at their sternal ends the line of demarcation may easily be demonstrated. I think that, based upon this examination, a general principle of localization may be stated, namely, that localization along nerve-trunks is usually less accurate than localization along the terminal portion of the nerves. The same general principle obtains in neuralgias. In intercostal neuralgia the painful spots of Valleix are found at the terminal bifurcation of the nerves, near the sternum, into superficial and deep branches.

So far, it is evident that at the level of the second thoracic vertebra there is something that is compressing the spinal cord. Is it something in the spine itself? Is it intradural? Is it a tumor of the spinal cord? Is it myelitis? If it were uncomplicated myelitis, there should be no root symptoms. The onset of root-pains in this case also means pressure, the result either of a tumor compressing the posterior nerve-roots or of an infectious granuloma or osteoma. If a tumor of the cord were present, the symptoms this patient shows might obtain; but, ordinarily, tumors of the spinal cord give a history of progressive, rather than intermittent, pain. In our opinion the radicular symptoms indicate either vertebral granuloma with pressure backward upon the nerve-roots, or extradural or possibly intradural tumor of the cord, but not myelitis.

An important point in the history, previously mentioned, is that the pain vanished promptly after its first appearance, not to return for some time. This fact indicates a varying degree of pressure. Such a varying degree of pressure has been described with *meningitis serosa circumscripta*, of which the first cases were reported within recent years. *Meningitis serosa circumscripta* causes signs quite similar to those arising from tumor of the cord or pressure myelitis.

We believe that in this case the pressure has started from the vertebra itself, as a tuberculoma, a sarcoma, or a syphiloma. The history, however, is absolutely negative for lues, and we do not admit the slightest probability of syphiloma. As to tuberculosis, there has been no fever at any time. His family physician confirms this statement. Moreover, there is no fixation of the vertebræ, and the tuberculin reaction is negative. What else might it be? A sarcoma, a fibroma, an osteoma, or *meningitis serosa circumscripta*. It is not possible definitely to diagnose, but from the symptomatology we feel that it is more likely *meningitis serosa circumscripta* than a fibroma. The absence of cachexia renders improbable a sarcoma. The skiagram apparently shows no osteoma, but bone pathology in the vertebral column often remains hidden. Of one thing we may be sure, that at the level of the second thoracic vertebra there is a pathologic process compressing the spinal cord, as signaled in this man's symptomatology.

An interesting symptom we forgot to mention is that sneezing occasions intolerable pain between the shoulders. It is obvious that the sneezing influences in some manner the pressure upon the spinal cord, probably by temporarily blocking the venous return.

[Dr. Murphy performed laminectomy in the upper thoracic region. He followed his usual technic, which was described in full in the CLINICS (February, 1914, p. 166; also June, 1915, p. 469). A median incision was made over the tips of the spinous processes, extending from the seventh cervical to the fifth thoracic vertebra. The muscles were then stripped back from the spinous and the transverse processes, and five of the spinous processes were removed, beginning below with the fourth thoracic, and passing upward to and including the eighth cervical. Only four, however, of the laminæ were removed. Venous oozing was encountered, as usual, in stripping away the muscles from their bony attachments.—ED.]

DR. MURPHY: This operation resembles the approach to the Gasserian ganglion in that at first the bleeding is superlative, but after packing and waiting a few moments it subsides. The bleeding in this case is mostly venous. In dissecting away the muscles one must work from below upward, following the erector spinæ group toward its median attachments, for if the dissection is made in the reverse direction, from above downward, the knife wanders to the side from above downward. We pack to stop the bleeding. . . . Now the transverse processes and laminæ of the vertebræ are exposed and we shall wait a few moments to stay the hemorrhage again. We bite off the laminæ with these large rongeur forceps, and discard the bone chips. I have never noticed weakness of the spine following the removal of the spinous processes or the laminæ, these structures being unnecessary to maintain the strength and stability of the spine. The disturbances of sensation here come just about the level of the patient's nipples.

During the past five or six weeks we have opened into the spinal canal several times. There is the dura. There is no pulsation of it now. We must go a little higher in the exposure so as to provide a free opening. One observes the second thoracic nerve coursing down here at the side of the dura. The resistance to pressure on the cord is like that of a stone wall: we cannot enter there, it is so solid and hard. See the difference! The left side is the worse.

We call attention to the difference between the two sides. The tumor is pressing laterally, particularly against the left side. We now free the growth on the right side: the dura is diseased. We have not freed the cord above yet: that is why we go higher before opening the dura. It is one constant succession here of advance and retreat. Now we are above the lesion, as the cord can be pressed forward without resistance. The length of the additional incision is about one and one-half inches.

We remove the laminæ of the second and first thoracic vertebræ. There is the neoplasm: whether it is in the cord or in front of it is impossible to determine until the dura is opened. Now the cord is free. At this spot there is great pressure from the bone, so that that piece of bone must come out. See the cord expanding and pulsating! It proves that we have relieved the pressure. This is an example of the decompression operation. We feel that decompression will prove more advantageous in the spinal cord itself than in the brain.

Now we are ready to open the dura, and the danger of infection increases as we enter the medullary zone. The cord sags above and below the tumor. The pressure bears against the cord in front. It is a solid bony tumor which, of course, we cannot take out. We are trying to decide if the growth is a granuloma: it feels like an osteoma. It is a growth on the posterior surface of the body of the vertebra—an osteoma pressing the cord back against the laminæ and bases of the spinous processes. The clinical course of this case emphasizes the same handicap we have had in every line of work, namely, procrastination. The longer the delay, the more pathology there is, and the less likelihood of complete restoration to normal. Decompression could have been performed upon this man in the early days just as readily as now. We are convinced that the next great book on surgery and on medicine will be on the subject of the "Morbidity and Mortality of Procrastination." Disease in many instances by timely treatment does but little harm—it is procrastination that destroys.

This operation results in complete decompression of the cord. It lets the cord expand backward by relieving it of pressure between the tumor in front and the laminæ behind. In tuberculosis it is not the curvature of the spine that creates mischief, but rather the granuloma that grows into the lumen of the spinal canal and presses upon the cord. Now the muscles and aponeurosis are accurately approximated, and we sew the aponeurosis across, obliterating the gap.

Let the record show that five spinous processes, beginning with the fourth thoracic and passing upward to and including the eighth cervical, and four laminæ were removed; that upon exposing the upper margin of the third spinous process we saw that the cord was being pressed back against the laminæ; that, after removing the second and first laminæ, we pass over the tumor and into a cavity free from compression above the tumor. The tumor was bony in type, being hard and non-pulsatile, and, therefore, not of the angiosarcomatous type that we encountered in a previous patient. (See CLINICS for August, 1913, p. 681.)

That patient had an angiosarcoma in this same region. We diagnosed the case as intraspinal angiosarcoma before operating, and gave a gloomy prognosis, but the patient and his family desired to have an operation performed. The man had hemiplegia. Upon exposing the growth by removal of the laminæ of the upper thoracic vertebræ, there was a geyser of blood and a great pulsating tumor. We closed the incision and the patient lived almost a year.

One must never insert drainage near the serous lining of the central nervous system. It is the same proposition as in a joint. See! That bleeding has stopped. We have not used any ligatures.

We think there will be restoration of all axons that are not yet destroyed, and many have escaped destruction. The axons already destroyed are out of commission permanently, because in the spinal cord itself axonal regeneration does not take place. Remember that within the true spinal cord nerve-axons have no neurilemma, and that absence of neurilemma means no regeneration. Down in the caudal zone, however, regeneration does occur, because after leaving the true spinal cord the axons acquire neurilemmas. All reputed cases of regeneration of the spinal cord after division—one reported early by Fowler and another later by Harte—were misinterpreted from an incorrect estimation of the extent to which the cord had been cut. In Harte's case it was fairly and clearly shown that but one-half of the cord had been cut, because the muscles controlled by the crossed pyramidal tract on one side, and those by the direct pyramidal tract, on the opposite side, were active, showing that below that point only half of the fibers were cut.

Let the record show that on opening the dura there appeared a circumscribed damming back of fluid, confined by the pressure above, which prevented its passing upward; that below the point of compression there was no pulsation until after the laminæ had been removed up to and including the first thoracic vertebra; that the faradic test of the zone below the point of compression was not made; that when the cord was displaced, this shining, bony tumor was revealed anteriorly; that this mass did not resemble a granuloma; that the dura was closed with catgut sutures, making an ectropion of the cut edges; and that the gap between the ends of the transverse processes was filled in with a muscular and aponeurotic flap.

[NOTE.—There was complete primary union and a material increase in motion and sensation before the patient left the hospital.—ED.]

There are two conditions of the central nervous system for which an early operation of decompression should be performed, and for which every practitioner should be alert. One is choked disc, resulting in impairment of vision. Choked disc is purely the sequence of excessive intracranial pressure. Furthermore, the optic nerve conveys axons which possess no neurilemma, and after the nerve atrophies, regeneration is impossible. *An aneurilemmic axon once atrophied is forever atrophied.* On the other hand, in the case of the peripheral nerves, as, for example, the median or the ulnar, perfect restoration of function may be secured by accurate contacting. The same is true of the brachial plexus. None of the nerves of special sense has a neurilemma; therefore, when these nerves *first* show evidence of intracranial pressure, decompression should be immediately performed, whether or not the cause or the localization of the pressure has been determined. Why? Because if the tumor be non-malignant, as, for example, a tuberculoma, which tends to self-limitation, the patient has been afforded an opportunity of cure before the onset of blindness. We had in the hospital at one time five patients with as many different degrees of impairment of vision from brain tumors, varying from slight impairment to complete blindness. All five patients lived. The one that was wholly blind remained wholly blind. The one that had but few axons destroyed remained with but few axons destroyed, but with practically normal vision. The other three retained the degree of optic atrophy that existed at the time of decompression, but they all lived. Each one could look back upon the doctor who had charge of the case before blindness developed and exclaim: "*If that man had been alert, awake, attentive, my blindness could have been averted!*" Such accusation is frightful. Imagine blindness overtaking yourself from somebody's oversight! In *that* spirit should one practise medicine.

The second indication for decompression is in the spinal cord. The moment paralysis appears is the psychologic moment to operate. Never mind whether you know the cause of the paralysis: go in and find out the cause before the patient becomes paraplegic. When the patients are in the superlative degree of paraplegia it avails naught to find out that the cause is a tuberculoma, an osteoma, or a fibroma. It is futile to remove the growth once there is degeneration of the nerve-fibers of the cord, permanently incapacitating and paralyzing the patient. We can frankly say to the patient that we do not know *what* is causing the paralysis, but that we do know there *is* a cause, and that now is the proper time to open into the spinal canal. When you utter your statements frankly, the patients are willing to consent. At first they hesitate, but if you tell them that blindness may and probably will develop and that paralysis may and probably will develop, they will permit you to do something to relieve the pressure upon the cord. That is the time to operate: do not let the paralysis develop slowly, but surely, and every day have the patient cry out with each increment of pressure: "Doctor! do something to relieve my pains!" *As day by day his vision becomes dimmer and dimmer, he pleads with his doctor for succor.* If it be a malignant lesion, the patient who is not operated upon dies. If it be a malignant lesion and decompression is performed, the patient retains the degree of vision to the end. Remember, the operation of decompression, whether of the brain or of the spinal cord, has a low mortality, being just as low as that of exploratory laparotomy.

In the spine it is not difficult to locate the level for decompression: one can usually come within one or two inches of the point. In the brain it is often difficult to localize the tumor, but one can at least localize it in the forebrain, the midbrain, or the hindbrain. Regardless of the site, the zone for decompression is the same, namely, under the temporal muscle (subtemporal decompression). When the tumor is situated posteriorly in the cerebellar zone, the point for decompression is constant—below the external occipital protuberance. By entering in this way an osteoplastic flap is unnecessary. Owing to less bleeding, decompression of the brain is much easier than of the spine.

TUBERCULOUS GRANULOMA OF VERTEBRÆ INVOLVING SPINAL CORD—LAMINECTOMY —INTERPOSITION OF MUSCLE-FLAP

Summary: Clinical history of case; analysis by Dr. Murphy; danger of direct attack upon a tuberculoma involving the cord; laminæ form points of counterpressure; tuberculoma compressing cord from before against laminæ behind; laminectomy, relieving point of counterpressure; consolidation of cancellous tissue of vertebræ; geniculate deformity of spinal cord; insertion of muscular flap to prevent contraction of tissues upon cord; prognosis as to function; value of the query "*Why?*"; hematoma hinders repair; elements composing a nerve—their anatomy, physiology, and influence upon regeneration; difference between suspension of motion and degeneration of a nerve; proper time to operate upon cord lesions; case of endothelioma of cord; postoperative treatment of tuberculosis; correlation of clinical history with operative findings.

HISTORY

THE patient, a male aged thirty-nine years, married, was admitted to hospital in August, 1914. The family history is negative. He had the ordinary diseases of childhood. Twelve years ago, in 1902, he had an attack of appendicitis, after operation for which he remained in hospital two weeks. Eleven years ago, in 1903, he had smallpox.

The present disease began eleven years ago, in 1903, when, twenty days after he was up from the smallpox, the right knee-joint suddenly failed him. There was no pain, but he could not walk. He was carried to bed. Three days later he was able to walk. A painless swelling appeared at the right knee-joint and extended about eight inches up the right thigh. It lasted twenty-four hours. Four days later he noticed a similar swelling on the volar surface of the left forearm, which lasted several hours. Five days later, while driving, a severe pain developed in the lumbar region and he lost control of both lower limbs. He went to bed, and remained three days. After getting up he felt another pain in the upper thoracic region, but of less severity.

In September, 1905, for the first time, he consulted a physician, who diagnosed Pott's disease and shortening of the left leg. A plaster jacket was applied to chest and abdomen for six weeks. The condition did not improve, severe intermittent pain appearing in both thighs, with perceptible loss of control of both limbs. Later a swelling developed in both groins, involving first the left side. From both swellings a quart of pus was aspirated. Aspiration was repeated 40 times, the last occasion being in June, 1906. In August, 1906, the patient was up and about, but walked with difficulty. The left leg was still short and abducted. A swelling which appeared over the left greater trochanter was likewise aspirated, but no pus was withdrawn. The patient remained in bed one year—from July 1, 1909, to July 1, 1910. He retained his usual weight. On getting up and about he still walked with difficulty, the left leg being still abducted.

Last month, July 15, 1914, numbness was felt in the toes of both feet, first in the right foot. The numbness ascended the limb, and by August had reached the level of the anterior costal margin.

Sensation to touch is abolished at no place. The right leg is flexed and cannot be extended normally, the left cannot be raised from the table. Motion of the toes is present. Since August 12, 1914, there has been loss of control of the sphincter ani. Six days later the bladder became uncontrollable. Rest in bed benefited both incontinent sphincters.

COMMENTS AND OPERATION

Dr. Murphy (September 2, 1914): This history is interesting in conjunction with two other spinal cases awaiting operation—a total of three spinal cases that present three different pathologic conditions and three different clinical courses. [These cases, grouped in this issue, afford an exceptional opportunity for the study of spinal surgery.]

Did Pott's disease precede the smallpox? Was the smallpox the exciting cause in increasing the manifestation of a tuberculous lesion that had been lurking in the posterior portion of the body of a vertebra, by liberating and freeing the focus, which occurs frequently in many of these cases? Or was the paralysis the sequence of an infection involving the spinal canal, metastatic to smallpox pustules as primary foci? The subsequent course is against its being a non-tuberculous metastatic infection, but in favor of its being a tuberculous, with acute edema and swelling of a granuloma inside of the canal suspending transmission. It did not destroy the spinal cord at all—merely suspended transmission.

In reviewing the subsequent course of that history, the formation of psoas abscesses, the fact that they were operated upon many times, and that they drained a long time, suggest that the lesion was tuberculosis of the spine, stirred up by the infection of smallpox. The clinical course, therefore, is that it was a tuberculous lesion; that it has penetrated through the posterior portion of the body of the vertebra into the canal, and that the granuloma is compressing the spinal cord; that now there is paraplegia, the result of this compression, and that this paraplegia is continuing and increasing, and in all probability will eventually destroy the nerve-fibers of the spinal cord unless the pressure be removed.

The next question is: What can be done to relieve this pressure on the cord, and by what means? By opening, curetting, and draining the granuloma? Yes and no. Remember, Sir Victor Horsley did that years ago, but when the profession adopted this treatment it found that opening of a tuberculous granuloma of the spine had the same untoward sequelæ as opening of a tuberculous granuloma elsewhere in the body—that a mixed infection often occurred, and destroyed not only the remaining portion of the body of the vertebra, but also occasionally the cord. Now our management of this case is to relieve the pressure produced by the granuloma penetrating the anterior wall of the canal and compressing the cord against the laminæ. Now, then, you cannot remove the body in front nor the granuloma in front, but you can relieve the counterpressure by removing the laminæ and letting the cord fall backward from the point of pressure.

There is a great deformity of the lower thoracic vertebræ (Fig. 181). We remove the laminæ and discard them permanently without impairing in the least degree the support of the spine.

From the protopathic and epicritic estimations of the area of disturbed sensation the seventh thoracic is the vertebra involved. The conformation here is altered by the chronicity of the disease. [Removes the laminæ and the spine of the sixth, seventh, and eighth thoracic vertebræ.] Now the cord is freely exposed. It is compressed from before and held behind, and is trying to protrude between the spinous processes. Here is the granuloma—a mass coming through from before and pushing the cord backward. The dura and nearby tissues are thickened. We do not wish to open the granuloma. It comes from the anterior wall and from the seventh thoracic vertebra, in accordance with the clinical calculation.

Let the record show that the cancellous tissue of the seventh thoracic vertebra is consolidated from this old inflammatory process, and from the additional weight it had to bear in its deformed state—an example of compensatory hypertrophy.

There is the deformity lower down—a geniculate deformity of the spinal cord. You can see from a distance how the cord bulges and how it dips between the laminæ as they recede from the spinous process. In this work one must heed the "*Safety first!*" slogan; any damage to the cord is irreparable, because the nerve-fibers of the cord, not possessing any neurilemma, cannot regenerate.

Now Sir Victor Horsley used to push the cord aside from its thickened point and incise and curette the granuloma. We do not do that. We feel that this patient is safer by not doing it. Why? Because the tendency of tuberculosis anywhere in the body is constantly toward resolution. Keep it away from mischief while it is resolving—keep it from mixed infection—and it heals. The mixed infection does the mischief in the lung as in the spinal column, it matters not what the locality.

We remove this spiculum of bone, lest it interfere with the spinal cord if the latter recede subsequently. What a beautiful exposure we have! what a splendid opportunity that cord has of coming back! We did not open the dura. Now we make a muscular flap to fill the space between these two bony processes, otherwise dense fibrous tissue will develop there. Being con-

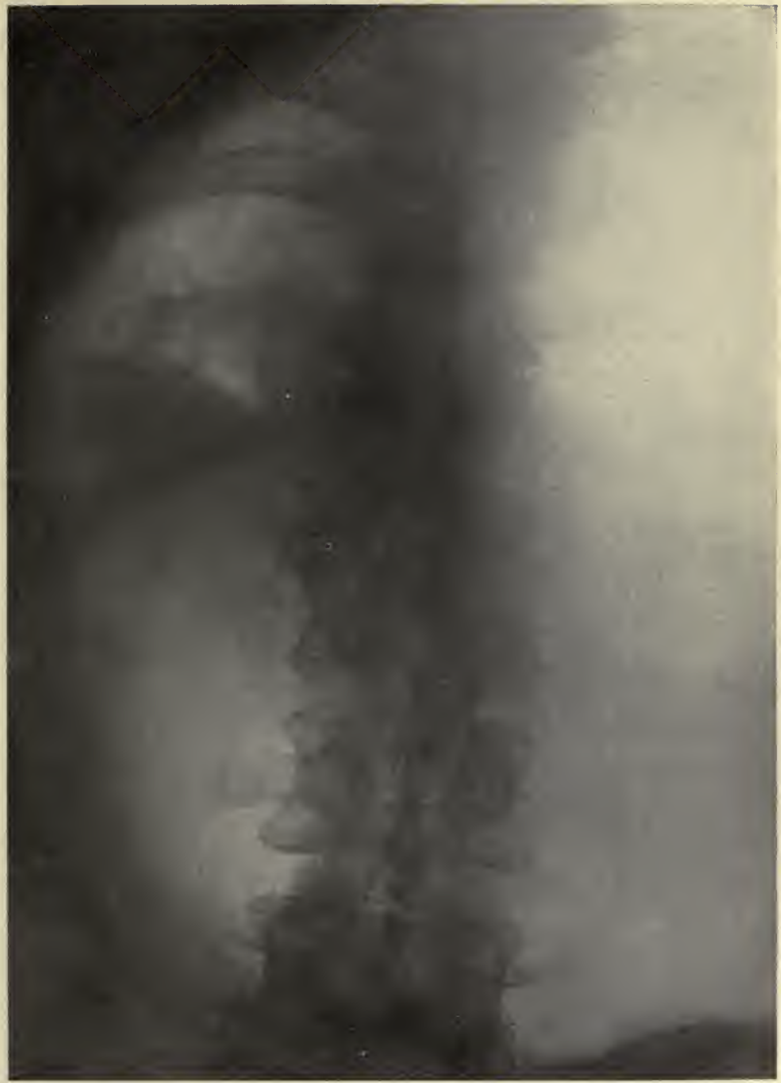

Fig. 197.—Pott's disease in lower thoracic region, and showing kyphosis and lumbar abscess.

nective tissue, it follows the law formulated by pathologists for years, observed by clinicians in the dawn of pathology, and expressed so beautifully in the words of dear old Professor Dudley Allen: "That connective tissue will consolidate and, consolidating, will contract." By inserting this flap and tacking down its edges, we supply a faithful agent for establishing normal tissue in between the ends of the laminæ and the spinous processes. We have been doing this for some time.

VISITING DOCTOR: What is the prognosis as to functional repair here?

DR. MURPHY: Favorable. Ask me the next question, just as I ask a student, "WHY?" That is why I preface "Why" to so many of these questions, because you merely believe what I say, but if I explain "why," you remember it—if I tell you "why," it is yours. If you merely have an expression from me it is always my property. I shall tell you why. The paralysis began only twenty-four days ago, and is not yet complete anywhere.

The bleeding has been controlled without ligating, as it usually may be in spinal cases, as in the brain. We insert a figure-of-8 stitch into the muscles so as to compress the entire area, leaving no recesses for the formation of a hematoma. A hematoma always hinders repair—around a bone-graft, a transplant, in the abdomen, or in the subcutaneous cellular tissue. A hematoma becomes a mass filled with granulations, and instead of obtaining primary repair of normal tissue, there occurs secondary union of cicatricial tissue. We close these layers much as we close the abdominal wall. Patients withstand these operations capitally, provided we avoid injuring the spinal cord high up.

On what does the prognosis of injury to the true spinal cord depend? What is meant by the true spinal cord? The portion above the conus medullaris. What difference does it make whether the injury be in the caudal zone or higher—in the true cord zone? The axons of all nerves consist of one or more of three elements. The essential element present in all is the axis-cylinder process; the next element is the medullary sheath, the third, the neurilemma. Be that axon in the brain, in the spinal cord, in the cauda equina, in a nerve of special sense, or in a peripheral nerve, these three elements must always be considered. They are of fundamental importance in one's clinical knowledge of the nervous system, particularly of the central nervous system.

Surgically speaking, the axis-cylinder is an elongation of the protoplasm of the cell-body. The medullary sheath is a coating for the axis-cylinder—the insulating material. It is also nutrient. The neurilemma is the regenerative sheath of the axon.

Now let us consider their functions. The function of the axis-cylinder is transmission: it is a wire of the telephone or telegraph cable. The medullary sheath is the insulating layer of the wires in the cable. The neurilemma is the mechanic that repairs the broken wire. Now, then, the presence of a neurilemma indicates potency of regeneration, and, as a corollary, absence of a neurilemma means impotency of regeneration. When a nerve is cut, the leading question in prognosis is—Has it a neurilemma or has it no neurilemma? If it has a neurilemma, it is capable of regeneration after approximation of the ends, just as the ends of a bone are capable of regeneration if contacted. *If the nerve has no neurilemma, even though it be approximated as early as five seconds after the injury, there will be no regeneration.*

Let us apply these truths. What peripheral nerves have no neurilemma? Just one set—the nerves of special sense—the optic, the olfactory, the gustatory, and the acoustic. Therefore, if the optic nerve be cut, it never regenerates; if the olfactory nerve be cut, it never regenerates; if the gustatory be cut, it never regenerates, and if the acoustic nerve be cut, it never regenerates. What other peripheral nerve has no neurilemma? None. Therefore, every other peripheral nerve is capable of regeneration if properly approximated.

Let us come back to the question concerning the spinal cord. In what portion of the spinal cord are the axons supplied by a neurilemma? In no portion of the true spinal cord. As soon as axons enter the true cord, they lose their neurilemma. The axon continues, the medullary sheath is retained, but the neurilemma is gone. Therefore, an axon that is cut in the true cord above the cauda equina never regenerates. If in the caudal zone, however, and the cut ends of the nerve be contacted, theoretically the nerve should regenerate; practically, we believe it does regenerate. The nerves in the caudal zone possess a neurilemma, a medullary sheath, and an axis-cylinder, just as the peripheral nerves, and they should regenerate under the same circumstances.

This patient is paralyzed. There is *suspension* of transmission. That means what? Not that the axis-cylinders are degenerated: they may be moderately compressed for weeks, more lightly compressed for months, before degenerating. *Operation for an injury to the true cord, when degenerated or when its axons have been cut, is futile, even though it be performed at the earliest possible moment.* If some of the axons have merely suspended transmission, and if we know from the responses that they are still living, we are certain that if the pressure be relieved, regeneration or restoration of function will occur, just as when, after compression of the ulnar nerve for one week, or two or three weeks, suspension of motion occurs, but returns within a short time after relief of the pressure. Suspension and degeneration are two different things: degeneration is irreparable; suspension always reparable after relief of pressure. Therefore the time to operate upon neoplasms involving the spinal cord is when first you notice suspension of transmission, for then you will benefit the patient; and the time not to operate is after you have determined that the neoplasm has destroyed the whole cord, for then you will not benefit the patient. Chronic pressure means irreparable degeneration; acute pressure of mild intensity means reparable suspension of transmission, and in many of these cases restoration occurs rapidly.

We have a case for to-morrow of interest in this connection, because the involvement is in the caudal zone. We have a case for operation on Saturday in the cervical zone. The latter patient has been in this hospital three times within four years. About a year and a half ago we removed an endothelioma of the spinal cord; the recurrence is not yet causing many symptoms. The patient says: "I can do chores around the house, but my spine is just commencing to annoy me." She is a judge's wife, and a very brilliant lady. From dealings with physicians she understands that if she waits until the spinal cord is degenerated, her paralysis will be permanent. (See next case.)

With relief of the pressure, this man should get well. We shall not treat the tuberculosis until the wound has been repaired, or until the end of three weeks. We institute treatment with graded hypodermic injections of tuberculin, and continue with hygienic measures. Tuberculosis is self-limiting.

Now we shall reconsider the course of the disease. The symptoms appeared three weeks after infection with smallpox. There was a sudden suspension of transmission which subsided within a few days. Then there was another suspension of transmission which also subsided. Then a kyphosis developed, then psoas abscesses, finally, a trochanteric abscess—manifestations, all, of spinal tuberculosis, which had been not only compressing the cord behind, but draining and suppurating before. The caries has been healed for years. The sinuses have been closed for years, yet he comes in with manifestations of a granuloma within the vertebral canal. Remember, the cord is rarely ever compressed by the angulation, but by the granuloma penetrating the anterior wall of the canal, elevating the periosteum as a sac, and compressing the cord back against the laminæ. Now you see why, if he has had but suspension of transmission, the prospects for complete restoration in the axons, notwithstanding the absence of neurilemmas, are good.

Let the record show that there was a granuloma in the body of the seventh thoracic vertebra compressing the cord backward against the laminæ; that there was extensive involvement of the bone above and below that point, the cancellous tissue being eburnated by the inflammatory process; that there was an acute angulation of the cord, a distinct knee formation, making at the ninth thoracic vertebra an angle of 40 degrees.

This area was soft, so that the cord was not compressed by the angulation. The pressure was higher up, at the site of the tuberculoma.

CONSTRICTION OF SPINAL CORD BY FIBROUS TISSUE FROM PREVIOUS OPERATION—RELEASE—DURAPLASTIC DECOMPRESSION

Summary: Postoperative history of case; comments and operation—dissection through cicatricial tissue, exposing cord; no neoplasm found—pressure due to constricting band of connective tissue; duraplastic decompression by inserting strip of fascia between cut edges of dura; fascia nourished by osmosis of plasma; danger of overlooking Horsley sponges in wound; endothelioma of cord surgically benign as concerns metastases, although pathologists differ; importance of figure-of-8 stitch in closing skin of back.

HISTORY (September 10, 1914)

[INTERN reads previous histories, which were published in the CLINICS of October, 1912, p. 695, and August, 1913, p. 733.]

Patient was readmitted to hospital in September, 1914, and recited the following history:

After the third operation, in May, 1913, the patient gradually improved for three weeks. The *x*-ray treatments were continued until July 15, 1914. During the last few months the numbness in the little finger and ulnar side of the ring-finger of the right hand has increased until now it involves the whole hand, and includes the radial border of the right forearm. The numbness has increased in the lower extremities also, especially in the right foot. At present the patient barely manages to get about the house, owing to the weakness in the knees. In walking, the left foot everts better than the right, and the steps are shorter. Flexion and extension are normal in the extremities. During the last few days she has noticed increasing difficulty with the right leg. Control of bladder and rectum is intact.

COMMENTS AND OPERATION

DR. MURPHY: At the first operation, four years ago, we split the fibers of the columns of Goll and Burdach in order to expose the length of the tumor within the spinal cord. The pressure symptoms have returned, but this time they indicate higher levels—the sixth, fifth, and fourth cervical segments—higher up than heretofore. The first time we removed just a part of the fourth cervical vertebra, but the tumor was only as large as a bean, and not so extensive. It was taken out earlier. Now read the microscopic findings.

INTERN: It was an endothelioma.

DR. MURPHY: [Incision and development of operative field.] You see there is no seventh cervical spinous process. Dr. Patrick has examined this case, and estimates that the site of pressure begins above, at about the eighth cervical segment this time. What spinous processes were removed in the last operation—from the third thoracic to the fourth cervical, were they not?

INTERN: Yes, doctor.

DR. MURPHY: Each time the histologic examination revealed endothelioma. This is the fourth operation upon this patient within five years. Now it is all cicatricial tissue which hides landmarks. First we must develop the outer wall, in order to gain access to the cord. Now we have exposed a bony landmark: it is the upper margin of the base of the spinous process of the third thoracic vertebra. This is much more difficult than the original operation. We have now reached the cord.

Let the record show that at the level of the first thoracic vertebra a fibrous band firmly constricts the cord. We have not yet opened the dura mater. We must first remove extensively this connective-tissue mass that overlies the tumor, in order to facilitate free opening of the dura and to lessen the amount of traumatizing and sponging of it. Owing to the universal presence of connective tissue in the field of operation, hemorrhage will be less than on the previous occasions.

Let the record show that the cord is exposed for three and one-half inches, and that there is a protrusion on the left of the cord at the level of the first thoracic vertebra. Whether it be due to a neoplasm or an osteoma cannot be determined until the dura is opened. Now the bleeding has ceased, and there is complete exposure of the spinal cord. As we open the dura the cerebrospinal fluid escapes.

Let the record show that the dura is thicker than normal, and that the cord pulsates.

Now we separate the pia mater from the cord at the spot where the connective tissue overlying the cord simulated a neoplasm. This was exactly the level at which Dr. Patrick localized the pressure, but we go higher to see if there is a recurrence of the neoplasm, and if that constricting band was the sole source of the pressure. There is good exposure of the cord for four inches.

Let the record show that no neoplasm is demonstrable: that the compression is caused by a transverse constricting band of connective tissue crossing the cord and uniting the bases of the laminæ of the first thoracic vertebra, and palpable before opening dura.

The patient reported early this time. To make a lasting decompression we must obtain a strip of fascia from the muscular aponeurosis and implant it between the edges of the divided dura. That follows the plan of duraplastic decompression of the cord described in one of the recent CLINICS. (February, 1914, p. 169; June, 1915, p. 469; see also a preceding case, p. 731.) This flap affords the cord a chance to spread backward as far as conditions demand. Fascia requires little or no vascularization, being nourished practically by osmosis of plasma, as in arthroplasty of the knee-joint. Owing to their small size, the Horsley sponges might be overlooked in the wound. Let the record show that Sister Victorine reports these sponges counted and correct.

The shock from this operation should be *nil*, for there was only division of cicatricial tissue, and the cord was not manipulated. A strip was interposed between the cut edges of the dura where the latter had contracted and compressed the cord.

Clinically, this tumor is benign, since it has been recurring without metastasizing, and has been harmful by virtue only of the compression it exerted upon the cord. Pathologists differ amongst themselves as regards the malignancy of these endotheliomata.

The skin is very dense and thick. The figure-of-8 stitch is important technically in closing the skin of the back, particularly in this zone of mobility and traction from the muscles of the upper extremity. In the preceding case we did not use the figure-of-8 stitch: the wound burst open, necessitating resuture.

Let the record show that there was no operative evidence of recurrence of the tumor; that the previous field of exposure for the removal of the tumor was easily demonstrable; that the cord rested in the normal position; that before opening the dura we felt a resisting mass, which proved to be a band of connective tissue crossing the cord and uniting the bases of the laminæ of the first thoracic vertebra.

TYPHOID SPONDYLITIS IN A TYPHOID CARRIER—CHOLECYSTOSTOMY AND APPENDICECTOMY

Summary: Clinical history of case; comments—typhoid spondylitis; positive Widal and blood-culture; the patient a typhoid-carrier, and therefore a menace to himself and to the community; appendix to be removed and gall-bladder drained, and autogenous vaccine to be prepared from bacilli grown on patient's own blood-serum; exposure and drainage of gall-bladder—cultures taken; appendix long, large, and deep, angulated and adherent—cultures taken; technic of appendicectomy; importance of careful ligation of stump to prevent postoperative bleeding; experiments of Dudgeon and Sargent upon bacteria in peritoneal fluid; early recognition of typhoid-carrier by detecting spondylitis; subsequent report.

HISTORY

The patient, a male aged sixteen years, was admitted to hospital October 15, 1914. He has had the usual diseases of childhood. Six months previously, in April, he had a slight attack of malaria. Five months previously, on May 15th, typhoid fever began. He was bedridden seven weeks, and was able to sit up the eighth and ninth weeks. The tenth and eleventh weeks he was up and about. His physician explained the seven weeks' confinement to bed by the presence of cholecystitis.

Five weeks ago, on September 8, 1914, while reclining, two hours after mealtime, acute pain, sharp and shooting, appeared in the lumbar region and radiated to the abdomen. The latter was distended, and the muscles contracted. There was fever, but chills, nausea, and vomiting were absent. This attack lasted one-half to one hour, and a second similar attack appeared six hours later, also two hours after a meal. Such attacks recurred several times a day until three weeks ago—September 25, 1914. There were night-sweats, and there was a fever of from 99° to 103° F. in the evening. The urine showed albumin, which soon disappeared. The bowels were constipated, the stools black and tarry. There was tenderness to pressure over the thoracicolumbar vertebræ, and moderate curvature of the spine. A plaster jacket extending from arms to hips was applied. At the end of two weeks it was removed, and the patient was confined to bed. The pain decreased, and the temperature fell to normal. The patient was then placed upon a canvas splint. There is no paralysis.

COMMENTS AND OPERATION

Dr. Murphy: This is the most interesting case of the day. He had sharp and severe pains, not a colic, in the upper abdomen. What has happened to him? He has developed a typical typhoid spondylitis—a stiff spine, not caries, since it does not cause destruction of bone: it is purely a joint inflammation. It involves also the peri-articular tissues, which thicken into a mass of connective tissue that eventually fixes the spine. The Widal reaction was positive, and, of greater importance, *Bacillus typhosus* was isolated from a blood-culture. He is, therefore, a typhoid carrier.

We know that the common lurking-places of the typhoid bacilli are the gall-bladder and the appendix. That is why the appendix will be removed and the gall-bladder drained—because he is a typhoid carrier. He is a source of danger to himself by developing a metastatic infection in the vertebral joints—a spondylitis. He menaces his community by scattering typhoid bacilli. We know that it is difficult to rid patients of *Bacillus typhosus*. He has become immune to vaccines. We shall prepare autogenous cultures from the bile, growing them upon his own blood-serum, for Rosenow has shown experimentally that by changing the medium the character of the microörganisms can be altered. Our logic is: Autogenous graft, autogenous vaccine: why not autogenous culture-medium? That is the way to get at it. We have already started the pathologist working upon that basis.

Here we make a small incision, our plan being merely to bring up the gall-bladder and drain it. The organ is not particularly white; it is not compressible. I aspirate the deepest stratum of the bile, because most of the microörganisms gravitate, like dregs. We pass a hemostatic stitch through the entire organ, unifying the drainage-tube and the gall-bladder. We make an entropion of the cut edges of the gall-bladder, turning them inward.

Let the record show that there was no macroscopic change in the gall-bladder; that while not so blue as normally, yet it was not altered enough to be called cholecystitis—at least not enough to produce a white color.

We make ectropion of the peritoneal edges.

Let the record show that Sister Ludwina reports the sponges counted and correct; also that, at the close of the operation, the surgeon inspected the field for sponges and instruments, but none was found.

Now we remove the appendix, approaching it through the outer margin of the right rectus muscle.

Let the record show that the visceral peritoneum is congested; that the tissues about the caput coli are also congested; that the terminal coil of ileum is thickened; that the appendix is greatly enlarged, angulated, and bound down deeply by adhesions; that it is six inches in length, and that its distal portion is infiltrated and dilated from continued retention of material under pressure.

There is no history, remember, of appendiceal obstruction. Now we have freed the adhesions: see how the appendix comes up! It should harbor *Bacillus typhosus* not only upon the mucosa, but in the muscularis. Cultural examinations will be made.

The appendix was removed in the usual way. There are three barriers to leakage—first, the ligature; second, the puckering purse-string, which is equivalent to a Lembert stitch; and third, the accurately applied Lembert stitch. Leakage at the mesenteric attachment has constituted the weak point in every intestinal closure from Billroth's time to the present. Now we cauterize that stump with a 95 per cent. solution of phenol, not neutralized. The stump is tied again. You will recall Hessert's splendid paper upon the bleeding that occurs from inverting the stump without ligating it. He reported a fatal case of his own, and collected several serious ones where bleeding into the lumen of the bowel occurred after inversion of the stump of the appendix without previous ligation.

The congestion of the peritoneum was due to the retention of the microörganisms in the appendix. In their experiments Dudgeon and Sargent showed that in the peritoneal fluid numerous lymphocytes are found, but seldom living microörganisms. We shall have the *tissues* examined for bacteria: while not in the fluid, they might still be in the tissues. The sterility of the fluid is no index of the sterility of the field. That has led to so many erroneous beliefs.

This is the first of this class of cases recorded where it was recognized early that the gall-bladder harbored *Bacillus typhosus*. What had attracted our attention was the typical typhoid spondylitis of which he complained upon admission, and which resulted from metastases from the gall-bladder as the primary focus.

Now we unite the cut sheaths of the rectus abdominis by means of a figure-of-8 suture. The muscle-fibers, uncut, fall back into place.

Let the record show that two test-tubes containing fluid from the pelvis were sent to Dr. Sweek for examinations and cultures, and that a piece of omentum was removed and bottled in a sterile test-tube for culture and staining.

SUBSEQUENT REPORT ON CASE OF TYPHOID SPONDYLITIS

DECEMBER 12, 1914: This boy came in with a typical typhoid spondylitis. The average case is treated in an orthopedic clinic as spondylitis (usually considered tuberculous). From the history we learned that he had had typhoid fever, and then concluded that he probably had typhoid spine. Then we went to work. We examined blood, urine, and feces, and all three yielded cultures of *Bacillus typhosus*. Then we performed laparotomy, drained the gall-bladder, and removed the appendix. The gall-bladder contents teemed with typhoid bacilli in pure culture. The appendix, of course, also harbored typhoid bacilli, these having been found in the feces. It was chronically inflamed and infiltrated, but he had never presented clinical signs of appendicitis. The presence of the typhoid bacilli made him a typhoid carrier, and caused the typhoid spondylitis. After the operation typhoid bacilli disappeared entirely from the blood. While found in the urine upon the second examination, they were absent from the urine and feces three weeks later at the third examination.

That is a striking illustration of how arthritic cases should be handled, and a search for the primary focus of infection made. The bile has been free from bacilli for a long time, so we are ready to remove the drainage-tube from the gall-bladder and let the patient go home. We feel that the discharge from the gall-bladder will not disturb him.

[Note.—The patient made an uninterrupted recovery.—Ed.]

CHOLELITHIASIS — ACUTE HEMORRHAGIC PANCREATITIS—INCISION AND DRAINAGE; —PANCREATIC CYST — DEFERRED MARSUPIALIZATION OF CYST

Summary: Clinical history of case—a classic picture of acute hemorrhagic pancreatitis sequential to cholecystitis; comments by Dr. Murphy—severity of pain in acute pancreatitis; primary attack cholecystitis, secondary, acute hemorrhagic pancreatitis; adhesions to be anticipated, owing to two previous infections; incision through left rectus, exposing cyst; cyst neither enucleated nor opened immediately, because of danger from trypsin; benefits of drainage; ectropion of peritoneum to prevent adhesions; importance of correct diagnosis of acute hemorrhagic pancreatitis—high leukocytosis—subnormal temperature; delayed marsupialization of cyst; brief note upon affections of pancreas.

HISTORY

The patient, a female, married, aged twenty-three years, was admitted to hospital May 8, 1914. The family and the previous histories are negative. She has borne two children, the elder being seven years of age. There were no miscarriages.

About two years ago, in July, 1912, twenty-two days after the birth of her second child, a sudden, severe colic developed in the right upper quadrant of the abdomen, and was followed by nausea and vomiting and belching. After enduring the pain seven hours, she summoned a physician, who prescribed an anodyne. Thereupon the pain gradually subsided. She has had several attacks in the same region, but none so severe, mostly lasting but an hour or two. The pain was always followed by marked nausea and repeated vomiting. There was never jaundice; never hematemesis; and she has never noticed blood in, or clay color of, the stools.

She had no attacks from December, 1913, until the present time—not for a period of five months. Four months ago, following an attack of colicky pain, her physician said that she was jaundiced, and diagnosed gall-stones.

As late as two days ago she felt quite well. Yesterday she was suddenly awakened by pain in the upper part of the abdomen, extending from the epigastrium to the right. A half-hour later the pain involved the whole abdomen. Within an hour of the onset the pain was prostrating and intolerable. No relief was afforded by change of position. She summoned a physician, who administered an anodyne, without, however, much mitigation. At the time of maximum pain she became nauseated, and shortly afterward vomited. There has been no bowel movement since two days, nor has she passed gas. Micturition has been scanty.

On admission the abdomen was tensely distended and universally tender: temperature, 96° F.; pulse, 126; respirations, 40. Leukocyte count, 27,200.

OPERATION

The patient was operated upon two hours after admission, at 10 o'clock at night, on Friday, May 8, 1914. A median incision was made. The abdomen contained much bloody fluid. The intestines were distended and intensely congested; slight peristaltic movements were noted. No obstruction was found. The uterus and adnexæ, as well as the appendix, were normal.

Upon examining the upper abdomen evidences of fat necrosis were seen in the omentum. The head of the pancreas was enlarged to the size of two fists: it was as hard as wood. The gall-bladder was compressible, but several stones were palpated in its neck. Free drainage was inserted. The operator, Dr. Golden, inspected the field on closing to see that no instruments or sponges remained, and Sister Victorine reported the sponges counted and correct.

SUBSEQUENT HISTORY

The patient was readmitted to hospital five months after the operation, on October 10, 1914. She had left the hospital two months previously, on August 15, 1914, having remained there for three months. She complained of bearing-down pains in the back, which radiated from beneath the left shoulder around the left side of the thorax to the left hypochondrium. No menses appeared between July 1st and September 24th. The pain continued, being more severe at night, when in bed. No further attacks of vomiting, chills, sweats, or attacks of jaundice have supervened. Constipation is a marked symptom. One night last week she noticed a swelling in the left hypochondrium, which was tender to deep pressure; she had also a slight fever.

COMMENTS AND OPERATION

DR. MURPHY: How severe was the pain? Where was the pain located this time, in differentiation from the pain of the first attack? The first pain was nearer the midline, beneath the costal angle. The next pain was *in* the midline, extending to the left costal margin. Note the difference between the *degree* of these two pains. The first pain was *colicky* in type. The second pain was *prostrating*. That description reflects fairly well the pathologic conditions present in both attacks. What day did the pain begin?

INTERN: The morning of the day previous to admission.

DR. MURPHY: She was admitted Friday night, thirty hours after the onset of the attack. The leukocyte count was 27,200, and the temperature subnormal. She had acute hemorrhagic pancreatitis, of which the history is a classic description. So severe is the pain that patients have to sit right down and will not let you move them. We remember a peddler out in Oak Park who had an attack of acute pancreatitis: he sat down on the curbstone in front of the drug-store and would not let any one touch him until the patrol wagon arrived and the police moved him from the doorway, so intense was the pain.

The primary attack of the patient before you was a gall-stone attack, and the secondary attack the fulminating hemorrhagic pancreatitis, which was operated upon and drained by Dr. Golden. Then she had a pericholecystic abscess, which we also opened and drained. Now she comes back with a sequence—a cyst of the pancreas, according to our diagnosis.

That is the regular course of this disease, and is typical in nearly all details. First, the pancreatic lesion is commonly the sequence of cholecystitis, calculous or non-calculous. Second, it is an acute fulminating lesion. *There is no lesion so severe in onset, none that causes such intense continuous pain or collapses the patient so much, as acute hemorrhagic pancreatitis.* It just goes right on paining each second a little more than it did the second before. The pain is enormously greater than that of perforated duodenal or gastric ulcer.

We now enter the abdomen through the left rectus muscle, because this cyst seems mostly in the tail of the pancreas. Now, you see, we have elevated the left rectus muscle in our approach to the cyst. Recall, she had two laparotomies, two infections in the upper abdomen, so we anticipate adhesions at this point. We are now in the free peritoneal cavity, which makes us feel comparatively happy—when working with the pancreas expressions should be but comparative.

We shall enter the lesser peritoneal cavity above or below the stomach, according to the displacement of the stomach. The stomach rests upon the cyst, which shines distinctly through the gastrohepatic omentum. In this case it is easier to approach the cyst through the gastrocolic than through the gastrohepatic omentum, because the stomach is high up in the field and cannot be displaced.

We now divide the gastrocolic omentum and expose the sac and then insert nosophen packing, which will not be disturbed until organic union has developed between this abdominal area and the anterior abdominal wall. The cyst is green in color. Shall we enucleate it? Judgment forbids: it is satisfactory to perform a complete operation, but never at the expense of additional hazard to the patient. The reason we do not wish to open immediately is the fear of soiling the abdominal cavity with trypsin, which has a deleterious effect upon neighboring structures. Were it of any other type than a pancreatic cyst, we could proceed less cautiously.

These cases progress admirably well when drained.

Let the record show that a collar of nosophen gauze was arranged around the exposed portion of the cyst and sutured to the abdominal wall, and that its identity was established by a safety-pin; also that a piece of gauze packing was placed in the center of this collar down to the wall of the cyst. This is to be removed at the time the cyst is opened without disturbing the encircling collar of gauze, which forms a coffer-dam to the peritoneal cavity.

We make an ectropion of the peritoneum so as to lessen the likelihood of adhesions in the lower zone, and to avoid fixation of the stomach to the anterior abdominal wall.

Let the record show that no sponges were inserted into the abdomen except the two packing sponges, and that Sister Victorine reported the sponges counted and correct. Let the record show further that before closure the operator inspected the field to see that no instruments or sponges were left in the abdomen.

Let the record show that there is a very large pancreatic cyst occupying the entire left quadrant of the abdomen and extending far to the right of the midline.

The important point in this case is the diagnosis, which must be made more from the history than from anything else.

The pain of an acute pancreatitis is so striking, so much in contrast with anything else, that you just naturally arrive at a diagnosis by the intensity of the symptoms. *The pain is prostrating: the attack, fulminating.* It is far worse than a biliary colic, worse than a renal colic, worse than a perforation. That history told the story very well.

You note that, notwithstanding the high leukocytosis, there was subnormal temperature. This is peculiar to cases of acute hemorrhagic pancreatitis, because the infection commonly throws out a blood-clot, which causes a rapid leukocytic response but no fever. At other times, however, a high fever is present. Drainage cures a very large percentage, but it must be continued for a prolonged period.

We shall let that packing remain until we are sure of a good organic union around the cyst, for three, four, or perhaps five days. Then we shall remove the central packing, open the cyst, and put in a drain, letting the cyst-wall collapse to keep the pancreatic secretion in a position favorable for immediate escape. The tube will not be disturbed for weeks—nay, months.

OCTOBER 20, 1914 (six days after operation): The central packing was removed, the cyst aspirated, and the packing renewed and fixed with a safety-pin.

Of pancreatic lesions, the commonest is a cyst following chronic pancreatitis associated with cholecystitis, calculous or non-calculous. Pancreatic lymphangitis, too, is doubtless of etiologic significance in infections of the pancreas. The next in frequency are neoplasms, benign or malignant, especially the adenocarcinomata. As long ago as 1884 Billroth ablated tumors of the pancreas. Recently an excellent paper by Finney has appeared upon the subject, and Dr. Coffey's experiments are very instructive. The cysts are the most promising lesions as regards feasibility of operation and permanency of cure. However, common to all pancreatic lesions is one source of great danger—the pancreatic juice, with its zymotic properties of producing fat-necrosis.

[NOTE.—The case progressed without untoward symptoms. The tube is still in. The secretion is gradually diminishing.—ED.]

[A previous case of pancreatic cyst, with discussion of the medical phase by Dr. Mix, was published in the CLINICS for April, 1914, pp. 247–260.—ED.]

FECAL FISTULA WITH CHRONIC RECURRENT APPENDICITIS—REMOVAL OF APPENDIX—CURE OF FISTULA

Summary: Clinical history of case; comments and operation—fourth day of attack many days too late for operation; remarks emphasizing complexity of fecal fistula operation; steps in technic for closure of fistula; removal of appendix; advantage of catgut over linen in presence of infection; review of steps of operation.

HISTORY

THE patient, a male aged thirty years, married, was admitted to hospital January 13, 1915, with a fecal fistula in the right iliac region. The family history is negative. He has had the usual diseases of childhood, and had malaria in 1902.

The present illness developed a year ago, February 5, 1914, with the first attack of appendicitis. A peri-appendiceal abscess formed. On the fourth day of attack the abscess was incised and drained, but the appendix was not removed. Five days after the operation, on February 14, 1914, postoperative hemorrhage arose from the deep epigastric artery. After controlling the hemorrhage, convalescence was uneventful until the appearance of a fecal fistula, which has persisted. The fistula discharges fluids and gases, but no fecal concretions.

The present attack began two weeks ago, on December 30, 1914, with sudden pain in the right iliac fossa, followed by nausea and vomiting, fever (101° F.), local tenderness, and ligneous rigidity. Since this attack the fistula has been more patent.

COMMENTS AND OPERATION

DR. MURPHY (January 14, 1915): How many days after the onset of the symptoms was the operation performed?

INTERN: Four days.

DR. MURPHY: He was not operated upon until *many* days after it began. He was operated upon *four* days after the onset of the symptoms. They did not take out the appendix. They did nothing but drain a peri-appendiceal abscess. That shows that *the fourth day after the onset of the symptoms is too late to operate for appendicitis*, and to expect the best percentage of good results, as by that time the pus will have passed beyond the wall of the appendix.

This fistula has the appearance of intestinal mucosa right up to the skin. As you contemplate operating upon this type of fecal fistula, you feel like imitating the late Christian Fenger preparatory to operation upon cervical tuberculous lymphadenitis. First he ate luncheon, then took a bath, then drew on his rubber suit, and then prepared to stay all day. We close this opening temporarily, so as to prevent welling forth of the intestines.

The first step in the technic is to prepare the operative field and then enter the free peritoneal cavity. The next step is to separate that portion of the intestinal tract containing the fistula from the anterior abdominal wall. If that cannot be done, the operative result bids fair to be unfavorable.

Let the record show that bleeding is profuse.

We can swing a finger entirely around the loop. We commence dissecting the fistula free from the outer side, just as we freed it a moment ago from the inner side. The fistulous tract is now entirely detached from the anterior abdominal wall down to the opening in the bowel.

Is the appendix still present, and if so, is it advisable to remove it to prevent another attack? It is our duty to remove it if it still exist. There is the large intestine and here the appendix. Here is where the abscess formed around the appendix, and probably involved by agglutination a number of intestinal coils simultaneously. Here is the communication between the intestine and the appendix, showing that the appendix continued draining into the small intestine. We separate the bond of union, making an entropion of the edge of the intestinal perforation by inserting and tying a puckering purse-string suture. We remove the appendix in the usual manner, inverting the stump with an accurately applied Lembert stitch. We touch the stump with a 95 per cent. solution of phenol, which is not neutralized.

Let the record show that the appendix communicated by a continuous lateral fistula with the small intestine, and was amputated in the usual way.

In closing the opening of the external fistula into the bowel we use chromicized gut because working in the presence of infection. Linen sutures inserted in the presence of infection may eventually have to be removed, otherwise the sinus persists months and months. We cover the line of union with omentum.

Let the record show that the fistulous opening involved a loop of ileum; that there was also a fistula between the small intestine and the appendix; that the appendix was contracted and still infiltrated and inflamed, not having recovered from the infection; that as the first step the free peritoneal cavity was opened to the median side of the fistula, enabling one to pass his fingers around the coil of intestine situated to the outer side; that the fistulous tract, together with bowel, was then dissected free from the abdominal wall and brought outside of the wound to the surface; that the fistulous tract was then freed from the bowel; that a Czerny-Lembert stitch closed the intestine; that the omentum was sealed over the line of union; and that, finally, the abdomen was closed without drainage.

Let the record show further that before closure the operator inspected the field to see that no sponges or instruments remained, and that Sister Ludwina reported the sponges counted and correct.

[Note.—There was complete primary union and the patient made an uneventful recovery.—Ed.]

(For a comprehensive discussion of the subject of fecal fistula see Clinics for February, 1915, pp. 1 to 38.)

PAPILLOMA OF BLADDER—SUPRAPUBIC CYSTOTOMY—RESECTION OF MUCOSA WITH CAUTERIZATION

Summary: Clinical history of case; comments and operation; causes of hematuria in order of frequency; the three-glass test for the source of hematuria; steps of operation; extensive papilloma on anterior wall of bladder—removal by excision of mucosa followed by cauterization and packing; importance of draining space of Retzius; postoperative discourse—characteristics of three-glass test in various localities; types of bleeding from neoplasms; danger of catheterization; treatment by fulguration does not fulfil expectations; siphonage as a means of keeping urine from field.

HISTORY

The patient, a male, single, aged sixty-three years, was admitted to hospital December 10, 1914. The family and past histories are negative.

The present malady began over two years ago, during the summer of 1912, with dull, burning, tingling pain that radiated from the glans penis along the perineum to the anus. The frequent micturitions aggravated the pain. Each micturition varied in quantity, and occasionally the urine was tinged with blood; at other times only cloudy. Pain and frequency were increased by coitus. Catheterization had never been performed.

Two months ago, on October 15, 1914, the patient could not urinate for one hour, although there was desire. With the help of medicine he shortly succeeded in passing urine containing blood, and during the following night passed blood several times. The next day he consulted a physician, who explored the bladder with a sound, with negative results. The urine was mixed with blood upon withdrawal.

On October 17th, two days after the temporary suspension of micturition, he went to a hospital. On the nineteenth he again passed blood. On the twenty-first he came to Chicago to consult a physician. He has never felt colicky pains, nor noticed gravel. Micturition closes with slight dribbling. There is no pyuria. Rectal examination reveals no enlargement of the prostate gland.

COMMENTS AND OPERATION

Dr. Murphy (December 16, 1914): What facts are lacking in that history? The description of the character of the mixture of blood with urine; the description of the stage of micturition in which the blood predominated, and, finally, the description of that stage in which there was practically pure blood. These are important factors in the differential diagnosis between the various lesions which cause hematuria. If you arrange in order of frequency the lesions that cause hematuria, you have first the lesions of the kidney; second, those of the bladder; third, of the ureter; fourth, of the urethra and prostate; fifth, the symptomatic manifestations of hemorrhage and of blood in the urine sequential to other diseases, the essential hemorrhages finally occupying sixth place. If you analyze micturitions in a definite way, you can reach some conclusions as to where this blood originates. How do you determine that?

Intern: By the character of the mixture.

Dr. Murphy: You collect the urine in three different jars—the first quarter, the middle half, and the last quarter in three different jars. That is the best gross clinical examination one can make in determining the character of the hemorrhage and its location.

In making the incision we enter through the linea alba, between the recti muscles above and the pyramidales below, and then divide and cut off the heads of the recti at their attachments to the symphysis pubis, thus gaining free access to the space of Retzius. We next push by gauze dissection the fat and the vesical reflection of parietal peritoneum upward, exposing the bladder and the end of the sound, which is palpable under my finger. The next step is insertion of the anchor stitch, to which we are still very much attached. This suture includes the entire thickness of the bladder and anchors it to the abdominal wall, so that in going in the second or fourth or twentieth time we shall not inadvertently enter the prevesical space. That is a plan we have followed for years. We open the bladder between these two stitches, revealing, upon retraction, the sound at the trigone (Fig. 198). Now those near can see the *fons et origo mali*, and understand why we detailed that history. See this huge papilloma protruding from the bladder! It is almost wholly to the left of the midline. We must excise the mucosa at this corner of the bladder, and then follow with the cautery. [This step is repeated upon the opposite side.]

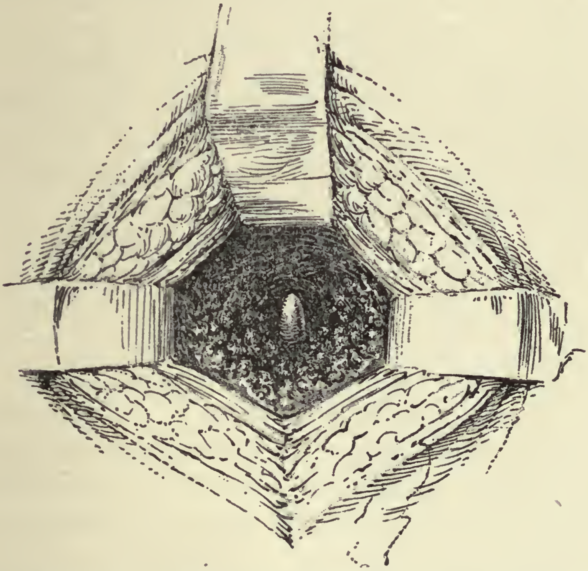

Fig. 198.—Papilloma involving entire base of bladder. Note tip of metal sound at internal urethral orifice amid the papillomatous area.

Let the record show that the papilloma had spread itself extensively over the anterior wall of the bladder.

After excising the mucosa we cauterized the place from which this tumor sprang, keeping clear of the ureters on either side, as they were plainly visible. Now the bleeding has been controlled.

There are the edges of the bladder up in the field, united as far as the foramen through which drainage emerges from the bladder. We want the bladder to compress the drainage tightly as a hemostatic measure, and prevent leakage of urine by conducting it from the field. Now we secure the bladder to the tube, higher up, so that if occasion arise to remove the tube before the catgut is absorbed, we can remove it out in the open. The figure-of-8 stitch anchors the bladder to the anterior abdominal wall, so as to diminish the dead space that would otherwise be left between the bladder and the wall.

Let the record show that Sister Ludwina reported the sponges counted and correct. Remember, on numerous occasions sponges have been left in the bladder and in the space of Retzius. We are equally careful of counting them here as in laparotomies.

We insert below the lowermost stitch a small tube into the space of Retzius to obviate poolings there. Retention in that space is an unfortunate complication because the space contains loose cellular tissue which rapidly necrotizes from urine, giving rise to a dangerous condition.

Taking up again the three-jar test: when the hemorrhage is from the kidney, the urine is uniformly tinted in all three jars. If it be severe, as at times from a calculus in the pelvis of the ureter, or as a symptom of non-ulcerative tuberculosis of the renal cortex, it may appear in clots, which often assume definite angleworm shapes. In these conditions the rule is that the urine is smoky in all three jars. When hemorrhage occurs from the ureter, it has practically the same characteristics as when it is from the bladder or from the kidney. Finally, when you come down to the bladder there is often clear blood in two glasses, with a middle zone of urine that may be comparatively free from blood. In symptomatic hemorrhage from the bladder—one in which tuberculosis elsewhere in the body causes an essential hemorrhage without ulceration—the blood may be mixed uniformly throughout the entire specimen. In bleeding typical of various growths, as here, or of polyps that extend into the *bas-fond* of the bladder, the urine is passed first with a gush of blood, then comparatively clear, and finally with a few drops of almost clear blood. That is characteristic. In hemorrhages arising outside of or anterior to the bladder, such as might be associated with lesions in the lower urethra, the blood appears independently of urination. By repeating this three-glass test three or four times you are enabled to reach a fairly accurate conclusion as to the location of the source of the hemorrhage, but not as to the etiologic factor. After you have finally located the source of hemorrhage, the diagnosis may easily be established.

In removing the tumor we exposed the mucosa, undermined it with curved scissors, elevated it, and then cut off the tumor, grasping the wall of the bladder as we progressed. After its removal we cauterized the area to check hemorrhage. We controlled the venous bleeding by the three sutures. By retaining the catheter there will be less pain and less danger. This tube in the space of Retzius will remain for ten or twelve days, until all the bleeding ceases, and until the granulations on the bladder have a firm foothold. While primary union of the wall of the bladder is frequent, yet there is always danger of infiltration of the urine into the fatty tissue, but here that danger is slight. Why? Because the patient has never been catheterized: he has been sounded, but not catheterized; therefore, we have practically sterile (non-infected) urine. He has a 38 per cent. renal efficiency, so the conditions are fairly good for a favorable result. The prognosis of papillomata is never good, because they recur and recur after removal, not only in their original position, but occasionally in the scar in the bladder and abdomen. We had hoped that, with the fulguration treatment, we would finally get better results, but thus far have obtained about equal results as with the open operation. The advantage of fulguration is that it obviates an open operation; that as soon as, by the initial manifestation of blood, the tumor is recognized as a small tumor, you can get the patient's consent to the treatment. This man has had hematuria nearly two years, and yet he permitted the papilloma to spread over that large area. Why was he not operated upon early?

After determining the location of the lesion in the bladder, we believe that early cystotomy is the proper line of procedure, and that it gives the best results.

The best method of keeping the field free of urine is by inserting a catheter through the lumen of the drainage-tube, attaching a siphon to it, and maintaining the siphon until we feel it is time to take out the larger tube and let the bladder close up.

It is exceptional to find a neoplasm in that situation. Neoplasms are more commonly met with below than above, and closer to one or other ureteral orifice.

[NOTE.—There was primary union of the bladder down to the drainage-tube. The case progressed favorably. There has been no recent report.—ED.]

(For an illustrated talk upon vesical papillomata by Dr. L. W. Bremerman, together with a classification of tumors of the bladder, see the CLINICS for April, 1914, pp. 337–354.)

FRACTURE OF LEFT PATELLA—OPEN REDUCTION AND WIRING

Summary: Clinical history of case; comments and operation—review of previous accident; results of conservative treatment poor; time to operate—cofferdamming of tissues by nature or by injections of formalin in glycerin; other questions preliminary to operation; plastic tendon operation for non-union after wiring; operation—fragments more movable than they seemed clinically; edges of aponeurosis between fragments the great cause of non-union; tear-fractures also found; technic of operation—importance of no-contacting method, especially of synovial membrane; importance of prolonged postoperative immobilization; postoperative comments—treatment of fissured fracture; fracture of both patellæ.

HISTORY

The patient, a male, aged thirty-eight years, married, was admitted to hospital February 15, 1915. The family and past histories are negative. At the age of seventeen, he had a Neisserian infection of short duration.

Sixteen months previously, on October 2, 1913, he was admitted to this hospital suffering from swelling, with slight tenderness, of the left knee-joint. The swelling extended from the middle of the thigh to just below the knee. This trouble began five weeks previous to admission, on August 30, 1913, when, while standing up in a motor traveling 15 miles an hour, the car suddenly swerved, throwing him out, and he struck his head upon the ground and the left leg sharply upon the fender. After the accident he made no attempt to bear weight upon the leg, and was brought immediately to hospital. A skiagram was taken, but showed no skeletal lesion. The patient was able to move the knee and ankle immediately after the injury, although with some pain. He also suffered with pain in the leg even when at rest. Three days after the injury a sodium silicate dressing was applied to the limb from ankle to middle of thigh. The patient lay in bed thereafter for ten days, and then was up for a time upon crutches. The dressing was left on for three weeks, and then removed. After its removal the patient noticed that a swelling was still present, which involved the knee-joint and extended four inches above the knee and two inches below. Before the dressing was reapplied the joint was aspirated, and bloody fluid withdrawn. The leukocyte count at that time was 12,000.

The patient had no chills at any time, and no fever, so far as he knows, although the temperature had not been taken after discharge from hospital. The swelling persisted in the knee for a long time, and subsequently extended farther down the leg. It was worse at night after having been upon the feet all day, but improved by morning after a night's rest. After persisting weeks, the swelling gradually disappeared, and from that time until the present injury occurred the knee has to all intents and purposes been normal.

The present trouble began the day of admission, on February 15, 1915, when his motor collided with a street-car, and with considerable violence. The injured knee struck the dashboard forcibly, and immediately intense pain manifested itself. He found himself unable to walk or stand, or even to extend the knee.

COMMENTS AND OPERATION

Dr. Murphy (February 20, 1915): When was the last accident?

Intern: Five days ago, on February 15, 1915.

Dr. Murphy: When was the first accident?

Intern: Over two and one-half years ago, on August 30, 1913.

Dr. Murphy: The first accident was what?

Intern: The patient fell out of a motor.

Dr. Murphy: No, he did not actually fall out. He was thrown forward and outward as the car suddenly swerved; but in saving himself his knee struck sharply against the fender. What did this first accident produce? Only a tearing injury of the posterior ligament of the left knee, with hemorrhage into the joint. The knee was hyperextended, tearing as well as twisting the posterior ligament and probably the crucial. The patient was in this hospital after the accident, and we had a good opportunity of observing him carefully. He was kept in bed ten days, and the limb dressed in full extension for ten or twelve weeks—that is, until we were certain that the torn posterior ligament of the joint had had plenty of time to reunite. Remember that it takes much longer for ligaments and tendons to unite firmly than for bone. There was no avulsion of the tibial spine by the crucial ligament in this case, which was perhaps a surprise to us, because the lesion is commonly caused by sudden, forcible hyperextension. Ultimately, the patient secured a perfect result.

Five days ago he was in another motor accident. This time he struck the same knee against the dashboard, and comes to us now with a more severe injury than the previous one was—a transverse fracture of the patella due to direct violence. You can see it there in the skiagram, which we have had placed in the shadow-box. Note the depression which is evident here in the skin over the anterior surface of the patella, where it dips in between the two patellar fragments. Since reaching the hospital the patient has been at rest on the back in bed, with a figure-of-8 adhesive plaster bandage over the knee and an ice-bag over that. If we should let him remain there with no further treatment than this, what would happen? In the next three or four weeks the space between the fragments would increase three-fourths of an inch, or perhaps more, owing to the contraction of the quadriceps muscle acting continually upon the upper fragment.

Just that slight but constant pulling of the quadriceps keeps the upper fragment far enough from the lower to prevent union. We now operate upon all cases of fracture of the patella. We believe that will be, or already is, the recognized procedure. There is a definite time for performing this operation, and it varies with the pathologic process present in the given case. In general, we never operate before the fifth day after the injury. Why? Because it takes anywhere from five to eight or nine or even ten days, depending upon the age of the patient and the severity of the injury, for the traumatic inflammatory reaction to become established in the knee. Wherever reaction to such injury occurs, a greater resistance to infection is developed, just as granulating wounds upon the surface of the body offer greater resistance than fresh wounds. The products of the reaction obstruct the lymphatics and fill the tissue spaces, thus greatly diminishing the danger of postoperative infection. If we must for some reason operate upon a knee in which there has been no recent inflammatory reaction, we inject the knee-joint with a 2 per cent. formalin in glycerin solution a week or ten days before operation, in order to produce an artificial chemical inflammatory reaction with consequent coffer-damming of the lymph-spaces and increased immunity to infection.

In this case nature has accomplished the same result by the blood which has been poured into the joint from the torn patella; but one must allow her the necessary time to act—five days at least. By that time the lymphatics and tissue spaces will have become plugged with the red blood-cells or the products of their disintegration, also with lymphocytes and serum, and the danger of infection ensuing after one has opened the joint and reunited the broken fragments of the patella becomes much remoter. A striking feature of the postoperative course of a knee-joint previously injected with the solution of formalin in glycerin is that there is not the subsequent effusion into the joint which one usually encounters after operating without preliminary injection. That is a prominent feature of these cases which is bound to impress the operator forcibly when he first uses the method. It is part of our routine preparation when we must operate upon a joint, the lymphatics of which we deem patent. We operate upon many such knees for loose or fractured cartilages, and always use this method of preparation unless we think that the recurrent traumas in the joint caused by the loose cartilage have already produced sufficient inflammatory reaction to coffer-dam the lymphatics.

Even after deciding to operate other questions arise—the direction of the fracture, the extent of comminution, the condition of the knee-joint, and the state of the quadriceps aponeurosis; also, the extent of separation of the fragments and of injury to the internal structures of the knee. Remember that while four-fifths of all patellar fractures are transverse in direction, the line of fracture is sometimes T- or Y-shaped, stellate, or longitudinal, and a few cases of frontal fracture, that is, a splitting of the bone into anterior and posterior halves, have been recorded. If the patella be badly comminuted, one must plan to contact the fragments by suture, otherwise early bony union is quite unlikely, Remember that the patella is a sesamoid bone, and, like other sesamoid bones, has relatively slight reparative and osteogenetic properties.

Fortunately, we need not depend entirely upon osteogenesis in this location. If the bone fail to unite properly, we may substitute a satisfactory tendon plastic through the use of the quadriceps tendon for this purpose. Make a transverse cut five-eighths or three-fourths of an inch in width and through the entire thickness of the tendon. From both ends of this incision split the quadriceps tendon down to its patellar attachment. Pass this strip of tendon over the patellar cleft and insert it into the ligamentum patellæ. Split the ligamentum patellæ so as to pass half of the quadriceps tendon flap through the center and over around each side, curling it around each half and uniting it end-to-end in front—a half-hitch loop giving a most secure type of fixation. Suture their cut ends in place as they lie, and also suture their point of intersection. By this method one has the quadriceps tendon as adequately and effectively joined to the ligamentum patellæ as it would be with an intact patella. One can even excise the patella completely, as we first did in a case about ten years ago, where it was seriously diseased, and substitute the quadriceps tendon for it in the way just described. That patient regained perfect use of the knee and walks fully as well as he did before the injury occurred.

[Dr. Murphy made his usual median longitudinal incision over the patella and, retracting the edges of the wound, exposed the line of fracture.]

There are the two fragments (Fig. 199, 1). They appear in this case to lie quite closely together. Notice how readily they slide by each other laterally as we move them, and how movable they are in reality, although before exposure they appeared relatively immovable. As we separate them and remove the clots with the forceps, we note a very interesting and important condition. It presents, we believe, the greatest possible obstacle to union and also the most frequent. We first called attention to it as long ago as 1886, when we had just such a case. That patient was a fireman who had been kicked by a horse. He was standing close to the animal, and the horse's shoe struck him an upward blow upon the knee. It divided the capsule of the patella, including the quadriceps aponeurosis. The aponeurosis was not only split, but also stripped up anteriorly on the patella, and a section of it insinuated itself between the two fragments and prevented union. That is exactly what happened in this case. It makes a very pretty demonstration. One can readily understand what influences the experienced surgeon to operate upon all his cases of fractured patella. See how absolutely impossible it would be for bony union to occur with this dense aponeurosis interposed between the two fragments! It would be as difficult to secure "bony ankylosis" of this patella while this strip of fascia is intervening as it would be for the patient with a fat-and-fascia-flap arthroplasty to develop a bony ankylosis with the flap still *in situ* between the joint-ends. We must, therefore, divide this portion of the patellar capsule and remove all interposing tissue. It is not enough merely to try to turn back this fascial strip, because it will not remain turned back. It must be excised to insure bony union. . . The vastus internus tendon has been turned inward a little here at the superior internal angle of the patella, and has torn away a small fragment of bone with it. This bony chip has dropped back into the joint, and we must remove it carefully; otherwise we shall certainly have arthritic changes develop afterward. Note how carefully we remove it, without touching the synovial membrane of the joint with either instruments or fingers. That care has a very definite and important purpose, which we shall speak of again. This fragment was so turned underneath the patella that it did not show in the skiagram.

Here is another portion—look!—of this prepatellar aponeurosis which has slipped in between the two bones from the distal side. The other piece slipped in, you remember, from above. This strip, too, must be excised, just as was the first. Unless one do that, see how impossible a sound bony union would be with three-fourths of the fractured surface of the patella covered with this dense fibrous membrane! [Excises it.] Here is another little fragment of bone which has dropped back into the joint from the distal strip of aponeurosis. We must remove it, too. Note, again, that thus far in the operation we have not touched a single spot inside of the joint with anything—instrument, hand, or sponge.

[Dr. Murphy then inserted the phosphor-bronze wire sutures, as shown in the accompanying illustrations.]

We have double-needled these phosphor-bronze wire sutures. The first needle is passed through the tendon of the patella below. It encircles the lower fragment of bone completely along its lower circumference, but remains everywhere within the tendon. The second needle is now passed through the quadriceps aponeurosis above the upper fragment of the patella in a similar way, and the two needles are brought out upon the same side of the bone (Fig. 199, 2). Now we are exactly repeating the procedure, except that we are starting from the opposite side of the patella with our double-needled suture (Fig. 199, 3).

These operative manipulations by the no-hand-contacting method are not easy, and remind one of trying to eat with chopsticks. In executing this joint work we are scrupulous about not touching anything in the field of operation with the gloved hand, and we have been just as scrupulous about not touching the synovial membrane with either the gloved hand, an instrument, or a sponge. Why? Because now there will be no effusion into the joint after operation, since nothing has touched the synovial membrane to irritate it. Never irritate the synovial membrane or in the least degree traumatize it in such a case if you can possibly avoid it. Whatever injury you may do to it accidentally will be paid for by the patient later, and paid for dearly, both in pain and in temporary disability.

Let the record show that there was found a transverse division of the patella; that there were present, in addition, two small bony fragments split one from each of the two patellar halves; that the deep portion of the aponeurosis of the upper patellar fragment was turned inward across the upper fractured surface, more upon its outer than inner side, and that the distal portion of the patellar aponeurosis caught the lower fragment from behind a little more upon its inner than outer surface, so that from two-thirds to three-fourths of the fractured surface of the patella was covered by these two fibrous strips, which would certainly have made bony union impossible if operative procedures had not been instituted. Let the record show also that these strips of fascia and the attached fragments of bone were excised completely without touching the synovial membrane of the joint with instrument, sponge, or gloved hand.

Let the record show further that two phosphor-bronze wire sutures were passed through the ligamentum patellæ and the quadriceps tendon, entirely encircling the two halves of the bone, one suture entering and emerging from the tendon on the right and the other on the left, so that we tied one on each side of the ligament (Fig. 199, 4); that the aponeurosis of the patella was then accurately adjusted on its anterior surface with such over-correction as was possible, in order to insure the firmest possible union (Fig. 199, 5); that the limb was then dressed in extension on a posterior, well-padded, wire-gauze splint.

In this case nature would have been powerless to have completed her task of bony union because these fractured edges were almost wholly covered with aponeurosis; and, in addition, if that were not enough, she had a sesamoid bone to deal with. We shall keep this limb completely immobilized for at least eight weeks. Experimental work as well as clinical experience has shown that sesamoid bones—the patella particularly—require complete, prolonged, and rigid immobilization to secure perfect union. Early mobilization is entirely contraindicated in these cases, not only because these sesamoid bones are slow to unite, but also because refracture is prone to occur with any ill-advised, early, or violent movement of the limb. Under this plan of treatment the outlook for the patient's regaining a perfect limb is most excellent. We expect some slight degree of stiffness of the joint when the leg is first taken from the splint and the patient starts to walk. But this early stiffness will disappear rapidly with further use of the limb.

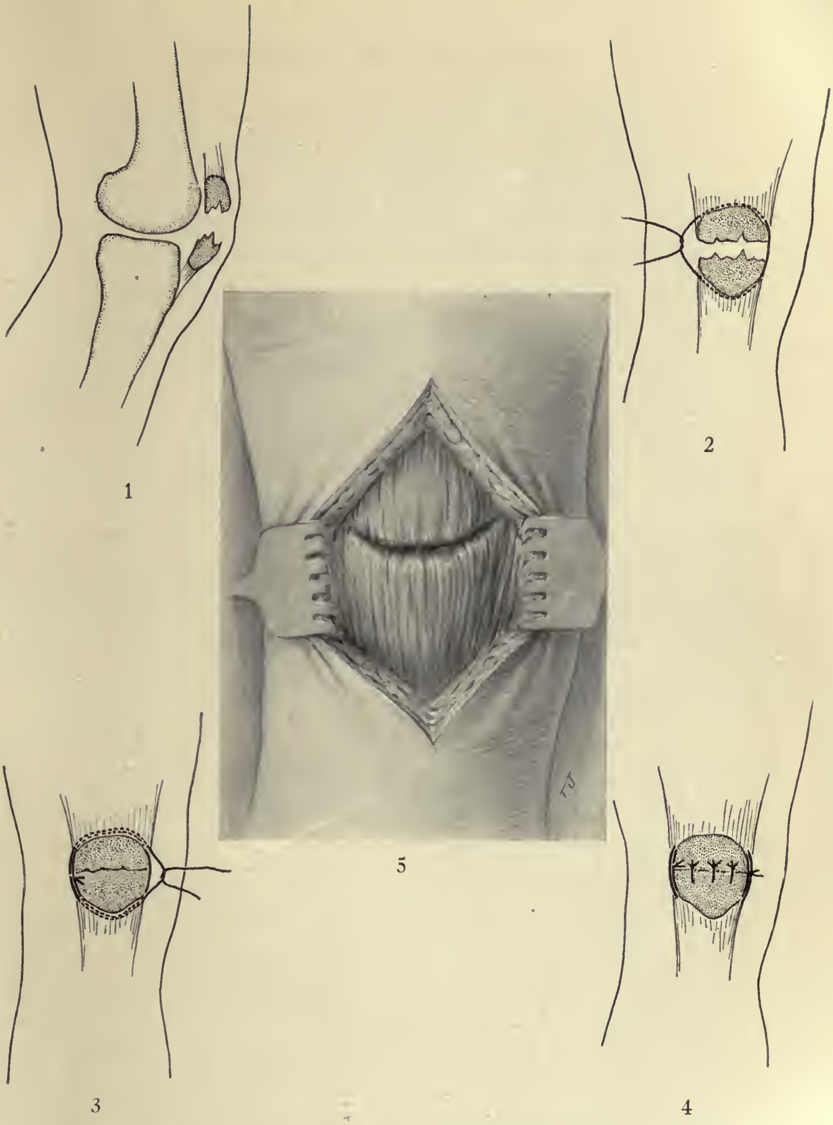

Fig. 199.—Fracture of patella—open reduction and wiring: 1, Shows extent of separation of fragments; 2, first phosphor-bronze suture inserted; 3, second phosphor-bronze suture inserted, the bone-fragments approximated, and the first suture tied; 4, anterior approximation sutures inserted into aponeurosis of patella—all sutures tied; 5, ends of torn quadriceps tendon approximated and turned in preparatory to suture. This provides a wide surface of contact between the ends, to insure early and firm union.

VISITING DOCTOR: Have you tried rubber sutures for encircling the patella in place of the wire?

DR. MURPHY: No, doctor, we have not. We think that an elastic suture in this location is directly contraindicated. Both experimental and clinical work have shown that sesamoid bones, including the patella, heal best with immobilization, and rubber sutures certainly would not give the required rigid immobilization of phosphor-bronze sutures to such a superlative degree. The rubber sutures would allow rubbing of the fragments upon each other, and that is exactly what we desire to avoid in this location. Remember that there is always considerable danger of refracture after old transverse breaks of the patella; and this tendency to refracture is most marked in the early weeks of renewed use of the leg, particularly following violent flexion movements. The presence of a strong, rigid reënforcement of the callus, such as the phosphor-bronze wire provides, is a great insurance against this dreaded complication.

VISITING DOCTOR: Do you operate upon all your cases of fractured patella?

DR. MURPHY: We operate upon all the cases in which there is demonstrable a space between the two fragments of the bone. In the fissured fractures, which we can now diagnose very readily with the *x*-ray, but which formerly we more frequently called sprains of the knee, operation is not necessary, because the bone-fragments are not separated, but with care and proper dressing remain in close apposition, and present no chance of fascia interposing, as in the case which we operated upon to-day. We keep the merely-fissured cases with a figure-of-8 adhesive plaster retention dressing applied to the front of the knee, and a well-padded posterior splint to the back, for the eight or ten weeks which are necessary to insure solid bony union.

Remember that both patellæ may be fractured simultaneously, just as both calcanei may be fractured in the same accident; and simultaneous fractures of the two patellæ are not so very infrequent—we should say they comprise from 1 to 2 per cent. Remember, too, that the same type of indirect injury which so frequently results in rupture of the patella may, in some cases, produce rupture of the quadriceps tendon above the patella, or of the ligamentum patellæ below it (we have one example of each of these lesions in the hospital at the present time), or, thirdly, may avulse the spine of the tibia. All these three other lesions produce symptoms somewhat similar to patellar fracture, and an equally serious disability. Unless one were aware of the possibility of their occurrence, he would not think of them, and the patient will not be given the benefit of the early operation, which is needed to secure complete functional repair.

[NOTE.—Complete primary union resulted; the patient was permitted to leave the hospital at the regular time with a circular gypsum case extending from gluteal fold to malleolus. He has had no pain or discomfort since. Flexion of the knee is increasing in range rapidly.]

FINAL RESULT

JULY 10, 1915: Patient now has flexion to right angles, has no pain or discomfort in the knee, and walks without a limp.

FRACTURE OF INTERNAL SEMILUNAR CARTILAGE—VILLOUS SYNOVITIS—EXCISION OF CARTILAGE

Summary: Clinical history of case; comments—analysis of history—prognosis unfavorable without operation; frequency of luxation of each cartilage; varieties of movable cartilages; rice bodies in knee-joint; poor results of conservative treatment; operation immediate in localities in which luxation is frequent; the three layers of the knee-joint capsule considered surgically; operation—villi found thickened and a cause of pain; cartilage hardened and fractured longitudinally—removed; fibrous capsule as the leather-bottle of the joint; no ligatures used; description of findings; postoperative discourse—resistance of coffer-dammed joint to infection; results good in absence of infection; postoperative treatment—importance of Buck's extension; pathologic changes in tuberculosis *vs.* pyogenic infection with differences in mode of repair; importance of relieving tension in inflammatory conditions; two sources of tension in an infected joint; vascular and synovial layers of capsule easily infected—fibrous layer and cartilage resist infection; infection in bone-ends.

HISTORY

The patient is a male, a physician aged fifty years, who was admitted to hospital May 10, 1915. The family and past histories are negative.

The present malady commenced three years previously, in 1912, when, upon turning suddenly to the right or left side after standing, he fell, hearing a cracking sound in the right knee, seemingly between the patella and the tuberosity of the tibia. This repeated itself about 20 different times. He had no pain when these cracking sensations occurred.

About two years previous to admission, on October 13, 1913, while attempting to jump over a fence, he experienced, at the instant of springing, an excruciating pain in the right knee. Although he managed to get up onto the fence, yet the intensity of the pain compelled him to rest on top of it. While resting there he noticed that he could not fully extend the leg owing to the severe pain. Flexion, however, was complete. After getting down from the fence he limped to the house with the right knee bent, walking upon the toes. After a night's rest the sharp pains were gone in the morning, but the knee remained sore, and was tender to pressure just beneath the edge of the patella: the limp persisted. The next day the doctor applied a short posterior wire splint, which he wore continuously for two weeks. Then he put on a longer one, which extended nine inches both above and below the knee-joint, maintaining the extension of the leg. This was left on for two months. After discarding it the knee pained and ached whenever he was on it long. Furthermore, a burning sensation began just under the lower and inner side of the patella, and has continued almost constantly since. Many local applications have been tried, but without avail. Then a circular gypsum case was applied and retained six weeks, followed by a posterior splint for another six weeks. It was now May, 1914, a year before admission: he was able to dispense with cane and crutch, and the leg did not pain when walking. He found, however, that he could not subject the foot to pressure, as against the pedal of a bicycle, without causing pain in the knee. Then he applied an elastic bandage. The knee was swollen at no time.

Ten months ago, on July 1, 1914, after walking up a steep hill, the knee began to ache. It has never locked since the time he was on the fence in 1913. Since July 1, 1914, the knee pained and ached whenever he walked much, or when he stubbed the foot or subjected it to pressure against an object.

COMMENTS AND OPERATION

Dr. Murphy: Which edge of the patella was involved?

Intern: I have not recorded that point.

Dr. Murphy: The inner edge. That is a fairly typical history, although not classic. You note, except upon one occasion, the absence of manifestations of locking that are so common with floating as well as with movable or fractured cartilages. You also note that the pain has been on the inner side of the ligamentum patellæ, extending to the internal tuberosity of the tibia and internal condyle of the femur: but little pain is referred to the last—it is practically all referred to the tuberosity of the tibia. Then, in addition, there was but slight effusion in the first instance, and, except upon one occasion, there was no severe pain. However, he has gradually become more and more incapacitated as a sequence of the recurrent traumata. Being a physician, he has watched it with great care from the beginning while treating it conservatively. Now, after that long period of time, he has finally concluded that the joint will be wrecked unless operated upon. We do not imply that a semilunar cartilage is going to be wrecked, because that is often wrecked in the primary injury. He would have engorgement of the vessels and thickening of the capsule and of the subpatellar bursa, so that the fatty bursa, interposed between the articular surfaces, would be pinched, in time becoming thicker and thicker, causing more and more trouble, until eventually the joint is destroyed by chronic traumatic inflammation without the supervention of hematogenous or metastatic synovitis.

With regard to the frequency of luxation of each semilunar cartilage, the ratio is about 50 times for the internal to once for the external. Rarely both are involved. We have had in our experience but one case of double luxation and both cartilages were free. Practically the same symptoms occur with movable or floating cartilages.

We divide movable cartilages first into the pedicled variety; second, the pinching variety, that shifts about into various positions; third, into the luxations and fractures—I always combine these two, because the condition that we regarded as luxation years ago is much more commonly fracture or detachment of an end of the cartilage. A simple luxation of the cartilage is rare. There is the type that forms pockets and develops elongated villi on the cartilage. Large villi may extend under the quadriceps tendon or ligamentum patellæ—they rarely go between the ends of the bones. There is also the type characterized by rice bodies, which develops thus: First, the rice bodies are elongated villi the size of a rice-grain, an oat-grain, or even larger; then, detached by the trauma, they become hard and polished. For a long time it was believed that these rice bodies were synonymous with tuberculosis of the joint. The late Dr. Fenger believed that in all cases rice bodies were tuberculous. We are now positive that they are not tuberculous in all cases.

This patient has a very severe traumatic synovitis, so that when we are through with the operation, we shall by no means be through with the treatment. The temporizing treatment by elastic knee-bands or by a gypsum case or other support, it is needless to say, has little or no effect in producing permanent cure. Once loosened, the rule is that these bodies remain loose until removed. So common is this that in localities in which luxation is frequent, as in the coal-mining districts of England, resort to immediate removal of the cartilage is the routine. The most conspicuous locality is the coal-mine at Newcastle, where the vein is so low that the men work in a stooped position. Immediately after the injury patients are taken to hospital, and as soon as the acute traumatic symptoms have subsided, the cartilage is removed and the men return to work within a few days. Sir Rutherford Morison showed us upward of 500 specimens removed by himself.

[Incision.] Now there is the fibrous capsule of the joint to which you have heard us refer so frequently: it is almost as dense and as strong as Poupart's ligament. Next is the place where metastatic infection lodges—the vascular layer sandwiched between the fibrous capsule superficially and the synovial membrane deeply. We free it separately, with the intention of plicating it and restoring its former position. This fibrous capsule is never the site of a primary metastatic lesion. Now we open the true capsule (Fig. 200, 1). Note the thickness of this vascular layer and the amount of fat it contains. We shall demonstrate what we mean by the villi that elongate and become pinched—the fatty type that becomes bruised, but never extends directly between the bone. There is one of the places "*where I get so much pain!*" as the patient expressed it. Here, under the bursa, is the thickening that has caused him so much trouble (Fig. 200, 2).

The cartilage is entirely loose at this angle and fractured longitudinally (Fig. 200, 5). See where it has become hardened!

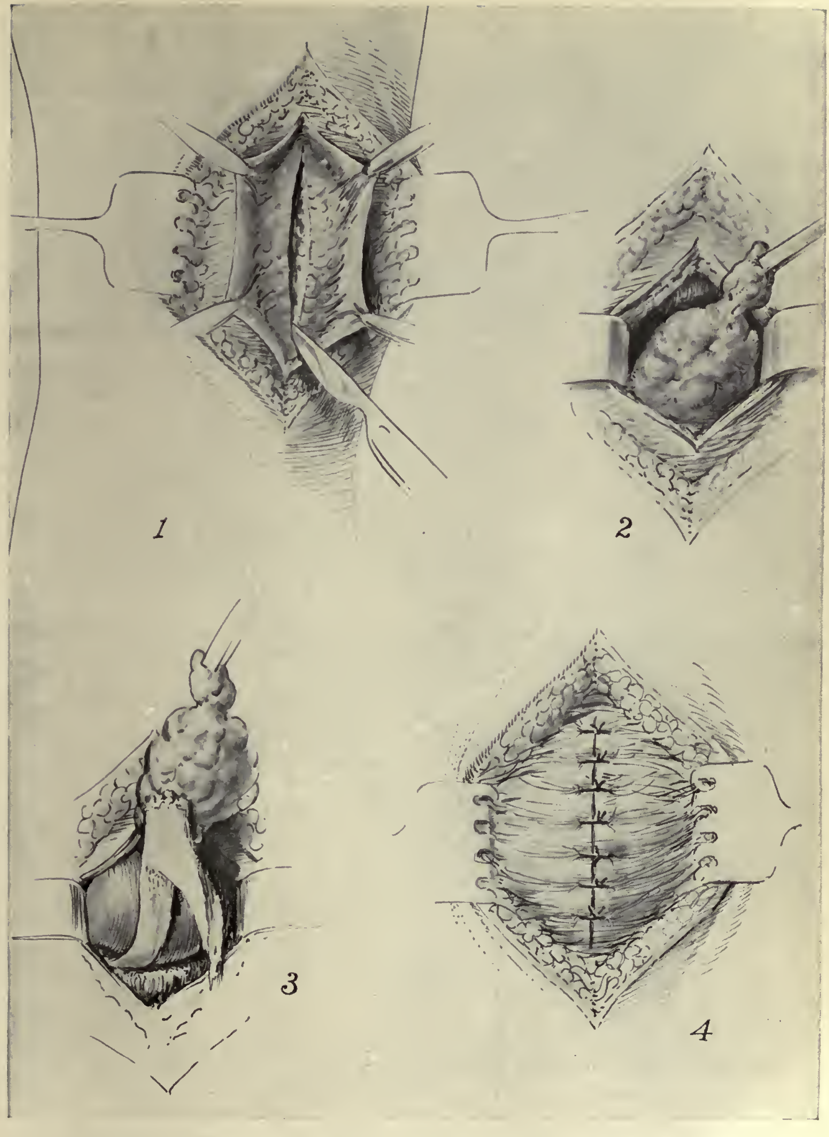

Fig. 200.—1, Fracture of internal semilunar cartilage. Longitudinal incision to inner side of patella. The knife is shown dividing successively the fibrous, the vascular, and the synovial layers of the capsule. 2, Retraction of edges of wound reveals inflamed and thickened subpatellar fatty bursa. Its inner margin is cicatricial from frequent pinching of its villi between the bones. 3, Upon delivering the fatty bursa the attached semilunar cartilage emerges at first unsplit, but soon bifurcating, the outer limb passing directly backward upon the upper surface of the tuberosity. 4, Interrupted sutures apposing edges of capsule.

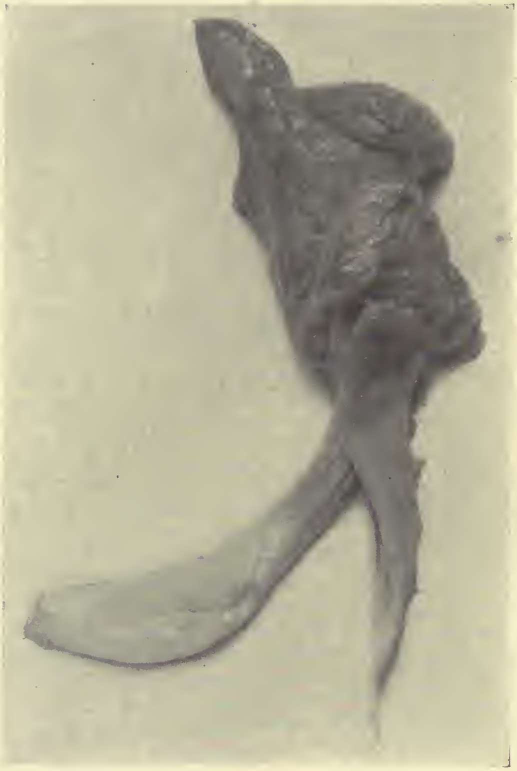

Fig. 201.—Pathologic specimen of fractured cartilage attached to hypertrophied fatty bursa. Outer limb of cartilage has been flattened and hardened from being caught between surfaces of joint.

Words have but little descriptive value compared with the force which seeing it carries from a teaching standpoint. You can see its two divisions: the outer portion is directed obliquely across the joint (Fig. 200, 3). Now we detach and remove the entire cartilage. You see how fruitless any type of extension or apparatus would have been.

Let the record show that the right internal semilunar cartilage was split from one-half an inch from the midline before to beyond the midline behind, or practically for its entire length.

We now suture the fibrous capsule back into position (Fig. 200, 4). It is the leather-bottle of the joint, and, in synovial infections, retains pus a long time, preventing the infection from escaping either into the joint or externally into the cellular tissue. It has a low degree of vascularity. This property of the fibrous capsule greatly enhances the danger of infection in the joint.

We practically never insert a ligature in these cases, but use this crushing hemostat, which compresses the tissues, thereby checking the hemorrhage. That oozing is coming from the line of suturing inside. The skin is closed with horsehair.

Let the record show that a pedicled portion of the elongated synovial layer of the capsule passed directly from the inner margin of the ligamentum patellæ close to where it crosses the joint; that the edge was flattened and hard from the repeated and continued bruisings; that it was freed, excised, and removed; that externally this pedicle was still attached to the inner angle of the internal semilunar cartilage, and after freeing this attachment it could be clearly seen that the cartilage had been split almost its entire length, from the inner side of the ligamentum patellæ before to within half an inch of its central attachment behind, opposite the spine of the tibia; that first the external portion was released from its attachment circumferentially, after which it could be seen that the central strip passed directly between the ends of the bones, spreading across the joint, and that this orientation of the fractured cartilage explained the futility of conservative treatment, and ultimately led him to seek relief at the operating table.

That is the most superlative split of the cartilage that we have seen: we call it a fracture of the cartilage. The danger of attacking this joint is naught compared with that of entering a joint not previously injured, since in this case the structures have been coffer-dammed by repeated inflammations from recurrent interarticular traumata, so that the joint no longer offers a fresh vaccination surface.

In these cases, in the absence of infection, the results are strikingly good. The patient will be kept in bed five weeks with a 20- to 30-pound Buck's extension applied, starting with 30 pounds and then coming down to 20, thus maintaining separation of the articular surfaces during the process of repair. The contracting muscles involuntarily compress these surfaces, and, if unrestrained, cause firm ankylosis. Furthermore, by keeping the joint surfaces apart, there will be practically no stiffness upon resuming use of the limb. Such is the treatment also for all metastatic and recent traumatic inflammatory conditions—extension and counterextension to separate the joint-surfaces, and never by the perfunctory application of a gypsum case. We general surgeons adopted the case because the orthopedists used it so much in treating tuberculous arthritides.

Comparison of the pathologic changes in the repair of tuberculosis with the pathologic changes in the repair of infection shows them to be two entirely different processes. Tuberculosis always repairs by cicatrization and encapsulation, never by phagocytosis. Infection always repairs by phagocytosis unless there is destruction of the endothelial covering of the joint: in that case it repairs by cicatrization and synechiæ, fibrous adhesions, and ankylosis. Note the difference!

In inflammatory conditions the indication is the prevention of pressure upon the inflamed zone. If you have a phlegmon in the forearm, how do you stop the destruction? By relieving the pus tension. If you have an inflammation in the joint, how do you stop the destruction? By relieving tension and applying extension. If a metastatic inflammation, you aspirate and inject formalin to sterilize the fluid within the joint. There are two sources of pressure in an infected joint, one resulting from the accumulated products of infection, the other from the involuntary muscular contraction which prevents motion during the stage of inflammation. If, during the latter stage, the joint surfaces be not separated, the cartilage is destroyed, and adhesions form, followed by ossification and resulting in ankylosis. This process is clear, and easily demonstrable.

There are but two tissues in the joint that are the seat of primary metastatic infection. One is the lymphatic zone between the fibrous capsule and the synovial endothelial surface, and the other the bone-ends. The cartilage is never the seat of a metastatic infection, owing to the absence of blood-vessels. The fibrous capsule is never the seat of an infection, metastatic or primary, except by contiguity. (A case similar in many respects to the above has been published in the CLINICS for June, 1913, p. 435.)

[NOTE.—There was primary healing. The case progressed without the slightest complication. The range of motion was as full when the patient left the hospital as it was preceding the operation.—ED.]

COMPOUND FRACTURE OF BOTH FEET: *RIGHT FOOT*—FRACTURE OF ASTRAGALUS AND OS CALCIS—EXCISION OF ASTRAGALUS—OSTEOTOMY OF TIBIA—INSERTION OF WEDGE BONE-GRAFT. *LEFT FOOT*—FRACTURE OF ASTRAGALUS, OS CALCIS, AND SCAPHOID—CUNEIFORM RESECTION OF TARSUS

Summary: Clinical history of case; comments—nature of injuries; available methods of correcting the deformities; details of operation upon each foot; citation of a similar case; results.

HISTORY

The patient, a male aged thirty-seven years, married, was admitted to hospital January 5, 1915. The family, past, and venereal histories are negative.

The present trouble began nine months ago, on April 3, 1914, when the patient was caught in a log-shoot and carried down the falls a distance of 200 feet at an angle of 45 degrees. In addition to concussion of the brain, his injuries consisted of fracture of ribs on the right side, with perforation of the lung and compound fractures of both feet. He was unconscious for ten days. On the tenth day the right foot became swollen and very painful: it was opened and drainage-tubes were inserted. The wound has continued discharging since. The left foot healed within about three weeks. At the end of three weeks both feet were dressed in gypsum cases, which were retained nine weeks—until July. At present the patient is unable to walk without crutches from the deformity of both feet.

COMMENTS AND OPERATION

Dr. Murphy (January 9, 1915): While shooting logs, this patient was caught between two of them, carried down the falls

200 feet, and lived to tell the tale! Both feet were injured, there having been a compound fracture of the astragalus and os calcis upon the right side, and a compound fracture-luxation of the astragalus upon the left side, with fracture of the os calcis and scaphoid. On the right side the astragalus has wholly disappeared, with the exception of a small fragment. How to correct that deformity and to make a walking machine of the foot is not so easy: it is mighty hard. We have been dreaming, as well as thinking about it. On the right side we could fracture the tibia and correct the deformity by inserting a bone-graft wedge. On the left side there is motion in the ankle-joint, but following the fracture the astragalus and os calcis were crushed and displaced inward, in which position they united and produced excessive callus. We apprehend that on this left side the tendons of the peroneus brevis and longus muscles have been avulsed, so that with the foot in an inverted position from contraction of the tendo Achillis, and with the excessive callus which has developed, we must devise a method of removing a wedge-shaped piece from the tarsus in an attempt to get a flat plantar surface. It is a hard task. We made plaster models of his feet to aid in devising a plan of attack. There is the position in which the man walks—upon the outer side of the left foot. With the exception of the luxation, the foot is normal. The tibialis posticus is contracted, and the tendo Achillis is contracted and deviated from the line in which it belongs. This contraction occurs a little to the inner side of the tibial malleolus, and swings the foot inward until the sole lies in a plane parallel with the long axis of the tibia (Fig. 202, 1). It will be hard to get this foot into a favorable position.

First Operation

We shall begin with the left foot. The backward displacement of the tibia cannot be overcome: it amounts to about half an inch. The circulation, in front and behind, comes from the external plantar artery, as well as from the internal calcanean branch of the internal plantar, and has been much interfered with. From the metatarsal heads to the ankle-joint the tarsus is one solid mass of bone. There is the fibular malleolus, and there

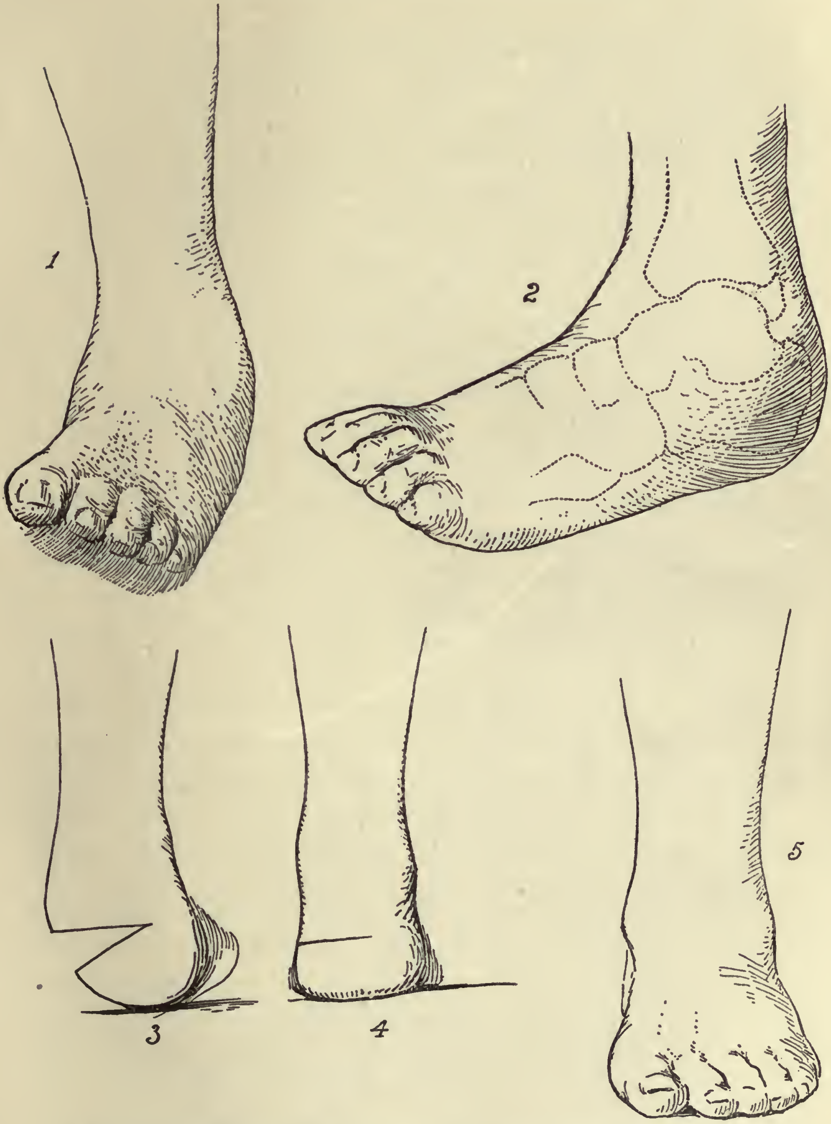

Fig. 202.—Diagrams showing deformity and correction, with plan of operation: 1, Ancient fracture of left astragalus, os calcis, and scaphoid, resulting in ankylosis of tarsus and inversion of foot (view from before). 2, Orientation of skeleton of foot with surface form (lateral view). 3, Removal of wedge from tarsus: base externally. External incision (posterior view). 4, Approximation of cut bone-surfaces, correcting the deformity of inversion. 5, Result: sole can be planted squarely upon ground (view from before).

the tendon of the peroneus longus muscle, passing down behind the base of the metatarsal bone of the fifth toe. You see it passes entirely across the sole of the foot to its insertion into the base of the metatarsal bone of the great toe. Now we remove a **V**-shaped piece of bone, thereby obtaining a flat plantar surface (Fig. 202, 3, 4, 5). There is the tendo Achillis, which we split. There is no danger of necrosis here. Now we drive in a 10-penny nail through the os calcis and astragalus into the tibia to hold it firmly with slight hyperabduction of the sole.

Let the record show that we succeeded in doing all that work without hand-contacting; that, after dividing the mass of newly formed bone in the left foot with the saw in a wedge-shaped manner, so as to free the antetarsus for rotation outward, we enlarged the wedge-shaped cavity, exposing the entire tibial malleolus, and then rotated the antetarsus outward.

Let the record show further that we did not have to insert a single ligature; that, after making the outward rotation, the flap was displaced sufficiently outward and a 10-penny nail driven through the astragalus up into the tibia so as to insure immobilization.

When you look at that foot it is in the corrected position of abduction instead of the former one of adduction, and it is well beyond the plumb-line (Fig. 202, 5). We did not reduce that posterior luxation, since, fortunately, we realized our limitations. It could not have been reduced. After an arthrodesis to produce ankylosis, always extend the gypsum case to above the knee, then immobilization is obtained *secundum artem.*

SECOND OPERATION

DR. MURPHY (January 23, 1915): We must remove the astragalus on the right side before we can correct the deformity. There is something in this right foot that prevents healing. We shall curette that sinus, since it is kept open by a sequestrum. The cavity will fill up rapidly. The remnant of the astragalus was found completely necrosed, and was excised.

Third Operation

In the right foot there was also inversion of the sole and eversion of the ankle (Fig. 203, 1). A portion of the necrosed astragalus was still in the cavity, accounting for the discharge which the intern described in the history. We opened it and excised the remnant of the dead astragalus. Now it is healed, and we shall correct the deformity enough to permit the foot to come flat. How much motion in the ankle-joint there is now after healing we have not determined.

We have under observation another patient who was thrown from a horse, receiving a luxation with a compound fracture of the astragalus and a displacement of the fibular malleolus outward. The foot was pushed up so that, as the man described it, the bone stuck out four inches on the outer side. We cleaned it, closed it, and it healed by primary union. The astragalus had become necrosed because the circulation had been cut off, and it was encapsulated without the formation of a sinus. It received but little circulation from the side, so that at the end of eight or ten weeks we reoperated and removed the necrosed astragalus. He had about the same type of deformity as this man.

In correcting that inversion deformity you must formulate your plans. You cannot supply a new astragalus nor change the articular surface of the tibia and the tarsal bones as they now stand; therefore the work must be done above the ankle-joint. That plan calls for osteotomy, followed by elongation of the tibia, so as to turn the lower end outward and let the plantar surface of the foot reach the ground. The patient cannot continue walking in that way. After dividing the tibia transversely we shall, if necessary, insert a wedge-shaped piece of bone from the other tibia (Fig. 203, 3, 4). We might take a piece of bone from the side of this same tibia by extending the incision upward, but we do not wish to remove it from a field contacting with the line of division. That is the scheme we have outlined. These are difficult cases to handle.

Let the record show that there is little or no motion at the ankle-joint, but that the motion is between the scaphoid and the os calcis, the astragalus having disappeared.

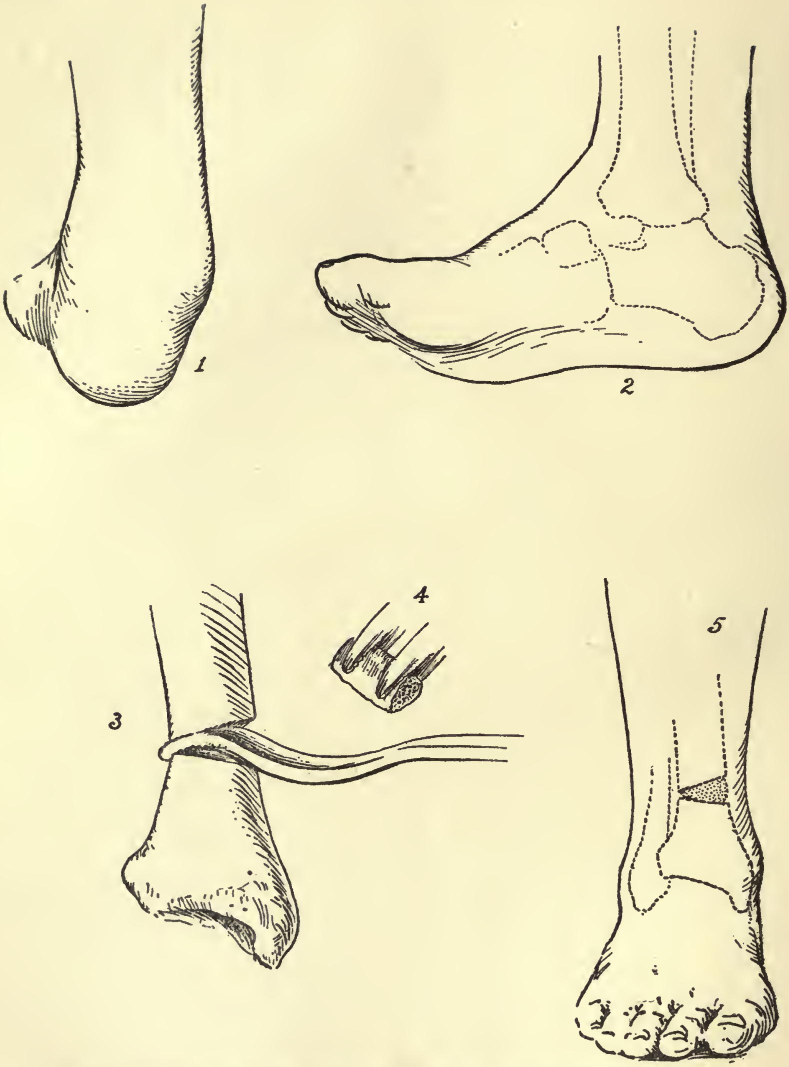

Fig. 203.—Diagrams showing deformity and correction, with plan of operation: 1, Ancient fracture-luxation of right astragalus, followed by necrosis and operative removal of this tarsal bone, and by inversion of foot. The ankle-joint was ankylosed. Postero-internal view of right foot. 2, Transparent sketch of foot showing absence of astragalus and tibia resting upon os calcis. 3, Osteotomy of tibia close to ankle-joint. Gap widened by lever into a cuneiform cavity with base internally. 4, Wedge-shaped bone-graft obtained from opposite (left) tibia. 5, Insertion of bone-graft wedge into cuneiform cavity of tibia, resulting in correction of inversion of foot.

There is the saphenous vein, ascending just to the inner side of the incision. We have elevated the periosteum. We shall obtain the wedge from the opposite tibia. In removing the graft make the incision over the internal surface of the tibia and then retract the flap. Now the graft is inserted and it stays right in position (Fig. 203, 5).

Now we remove the constrictor. The bleeding is from the vein. We insert this suture parallel to the long axis of the vein and of the wound, so as to relieve tension from the cutaneous stitch.

Let the record show that we divided the right tibia about an inch above the articular level, spread it, and pushed it down about five-eighths of an inch; that we then removed from the opposite tibia a wedge-shaped fragment of bone to fill in the gap; that after insertion it adjusted itself and retained the tibia in good position; and that the foot was dressed in a gypsum case.

Under favorable conditions the dressing will not be disturbed for four or five weeks. Remember, this horsehair may stay in six or eight weeks without doing harm.

Now we shall remove the nail from the left foot. That blood is from the hematoma beneath the skin. We aspirate the fluid in the left ankle-joint, and send it to the pathologist for examination.

[NOTE.—There was primary union. The patient was able to walk on both feet with both soles flat upon the floor, without the aid of cane or crutch. The result was far beyond our anticipations.—ED.]

RICHMOND STEPHENS, M. D.

THE CLINICS
OF
JOHN B. MURPHY*

Volume IV Number 5

CARCINOMA OF GUM AND OF SUBMAXILLARY LYMPH-NODES—EXCISION OF CANCER-BEARING AREA

Summary: Clinical history of patient; comments—leukoplakia buccalis; development and clinical course of the neoplasm; fatal prognosis of cancer of mucosa; wide-spread leukoplakia in mouth; early removal of isolated patches of leukoplakia indicated; difference between cancer of mucous membrane and of skin; radium *vs.* operation; effect of trauma upon a mole—illustrative case; necessity of wider excision of mole than of cancer; citation of a very early cancer of lip; operative findings; steps of operation; points emphasized.

HISTORY

The patient, a male aged sixty-three years, was admitted to hospital September 18, 1914. The family history reveals that one brother died of cancer of the stomach. At twenty years of age the patient had an attack of pneumonia of two weeks' duration.

The patient has been a tobacco-chewer since he was eighteen years of age. Three years ago, in 1911, he noticed at the spot where the quid lodged, between the left cheek and the adjacent lower gum, that the mucous membrane had a grayish-white appearance, and, three or four months later, that the area was enlarging and becoming elevated; it was somewhat painful at that time. A physician said that he had a "tobacco-sore" and advised him to discontinue the use of the weed. He stopped chewing and used a mouth-wash, and while this allayed the irritation, yet the ulcer gradually enlarged.

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*This number of the Clinics was edited by Dr. P. G. Skillern, Jr., of Philadelphia.

In the August number of the Clinics, on p. 579, owing to a printer's error, *crystals of argyrol* was given as chrysolate of argyrol: of course, there is no such preparation.

On p. 581 of the same number, in speaking of sodium cacodylate, "two grains once a week" should read "two grains the first day."

Sodium cacodylate administration is described in detail in this issue (p. 953).

The patient was in the Cook County Hospital for about one year and was given local treatment—a wash only. The ulcer-area kept gradually enlarging, became indurated, and showed cauliflower-like excrescences from which small bits of yellowish-white tissue were exfoliated.

While in the Cook County Hospital the patient had the teeth of the lower jaw, from the canine to the ramus, extracted in the hope of obtaining relief from the pain, but this procedure did not ease him. Since then the pain has been more constant and the growth has enlarged, extending from the socket of the canine tooth to the ramus, and involving the mucous membrane along the gum and along the margin of the cheek near by. There is much induration, but there are no palpable lymph-nodes in the neck. There is no history of gross trauma.

COMMENTS

Dr. Murphy (September 21, 1914): That papilloma-formation was the first deviation from the leukoplakia. A leukoplakia is a displacement of the epithelial cells covering the mucous surface plus a deposit of connective tissue, and therefore it is usually associated with some external irritation. Then comes hyperplasia of the normal cells as an epithelioma developing in or around the patch of leukoplakia. What did he have first?

Intern: He had first a whitish discoloration.

Dr. Murphy: Called what?

Intern: Leukoplakia.

Dr. Murphy: Second?

Intern: Cauliflower-like excrescences.

Dr. Murphy: A papilloma formed around the patch of leukoplakia and discharged fragments until it presented as an ulcerating, cauliflower-like induration of the neighboring tissues. The story is probably a little shorter than the average story because the patient had stopped chewing tobacco for so long. He had always kept the quid between the cheek and the gum of the lower teeth, in the buccal cavity. This little pouch underwent changes in appearance, a leukoplakia developed, and finally metamorphosed into one of the most malignant types of carcinoma, namely, carcinoma of the mucosa. Aside from the melanotic carcinomata, this is the most malignant type we have: though it is often slow of growth, yet it is very malignant ultimately. After examining him we said we would try to remove all the growth by excision of the mandible, if necessary. His son asked: "What is the hope of a permanent cure?" We replied that he will not have a permanent cure because he is afflicted with carcinoma beginning in the mucous membrane, which is usually fatal. There are exceptions, but they are few. The proper time for treatment was when that patch of leukoplakia was first noticed.

Occasionally one finds these patches of leukoplakia over the whole tongue, and lining the cheeks and the entire mouth. This gross type rarely results seriously unless subjected to local irritation. Where you find a circumscribed patch of leukoplakia at the angle of the jaw from heaping-up of the cells of the mucosa always ablate it before it becomes carcinomatous, as it so frequently does. If you find local leukoplakia on the tongue or cheek from chronic irritation of a tooth or a dental plate, remove it early, the moment it acquires that whitish color, which indicates that the next step will be thickening and then increased ulceration—the beginnings of malignancy.

In the management of the areas of leukoplakia and of these carcinomata of the mucous membrane we must employ different methods from those suitable for the epithelial growths that involve the skin. Some that develop upon the outer portion of the lip respond fairly rapidly to *x*-rays and more rapidly to radium. That is the consensus of opinion in this country with men of the greatest experience in ray-therapy. Six months ago we referred a patient with a lesion of this type to Dr. Robert Abbe, of New York, for radium treatment, but Dr. Abbe would not touch him, and sent him back to me for operation. Dr. Abbe's estimate corresponds exactly with the statement made to me last summer by Captain French, chief of the Radium Institute of London, who said that radium has no value in these cases of carcinoma of the mucous membrane of the mouth, but that operation should be performed. And yet these patients are permitted to reach the advanced stage of this case of cancer before being operated upon!

The proposition here is that, when first recognized both by the patient and by the physician, the lesion was only a leukoplakia. The future method of dealing with cancer will be the excision of precancerous lesions—the removal of tissue which might become exceedingly malignant, as, for example, melanotic warts or melanotic birthmarks. Quiescent year after year, the day eventually dawns when the little pimple is traumatized and suddenly commences to spread and disseminate throughout the entire body—usually in the submucous or subdermal zones—terminating, according to rule, fatally.

Recently a patient of ours traumatized a little mole down on his side one morning at exercise. Two or three weeks later the mole began to appear red and angry. The physician who first excised it made too close an incision. When he came in to us he had thousands of black spots beneath the skin and spread over the body. What lesson does this teach? The removal of a pigmented mole early, always excising a wide area, the mole being much more dangerous than an ordinary sarcoma, since it is transmitted through the circulation and not through the lymph-spaces, as carcinoma most frequently is.

[A case in which the scapula was involved by sarcoma after a mole over its spine had been traumatized was published in the CLINICS for June, 1913, p. 521.]

One week ago to-day we operated upon a man for a small lesion on the lip, not so large as a split French pea—not more than one-sixth of an inch in diameter. It seemed the most superficial manifestation which could be labeled carcinoma. He had had it six months. After excision the pathologist examined it and reported squamous-cell carcinoma.

[A discussion of squamous *vs.* adenocarcinoma of the lip was published in the CLINICS for August, 1915, p. 611.]

OPERATION

Findings.—Four enlarged submaxillary lymph-nodes, the largest being, as usual, that embedded in the anterior pole of the submaxillary salivary gland. The mandible was smooth and uninvaded by the cancer.

Steps.—Incision for ablation of enlarged submaxillary lymph-nodes; ligation of facial artery; preservation of uninvolved margin of lip; removal of periosteum; removal of involved mucosa from side of tongue, suturing remainder of lingual mucosa to that of cheek; pedicled flap for reconstruction of chin outlined below; stitch inserted to relieve flap of tension; gutta-percha drain.

Points Emphasized.—The greatest operative danger is from bleeding into the mouth; necessity of preserving mucous lining of floor of mouth, otherwise the patient is in a very uncomfortable situation; tension upon flap hazards its circulation; had the disease involved the bone, the latter would have been resected and a metallic substitute for the mandible inserted, since the region of the jaws is practically the only part of the body where a foreign body will heal and remain in place in the presence of infection.

[In association with this case it is of interest to review the case of "Malignant Epulis of the Mandible," which, with illustrations of several tumors of the mandible, was published in the Clinics for June, 1915, pp. 567–577.]

CARCINOMA OF TONGUE AND OF SUBMAXILLARY LYMPH-NODES—AMPUTATION OF TONGUE AND EXCISION OF CANCER-BEARING AREA

Summary: Clinical history of patient; comments—significance of ulcer on side of tongue in a man of fifty; diagnosis of such an ulcer; cauterization obscures picture of disease; delay in diagnosis probably fatal in this case; sins of omission worse than those of commission; high mortality after excision of entire tongue; comparative percentages of cancer-mortality with and without lymph-node metastases; operation in this late stage to induce euthanasia; diagnosis established by examination of a frozen section; the country doctor a better diagnostician than the average city doctor; operative findings; steps of operation; points emphasized during operation.

HISTORY

The patient, a male aged fifty-one years, was admitted to hospital June 11, 1914. The family history is negative concerning carcinoma. Twenty-three years ago, in 1891, he had a sore upon the penis which lasted only three days and was not followed by an eruption.

Seven months ago, in November, 1913, while being treated for gall-bladder trouble,—jaundice, pain over the gall-bladder, but no colic or vomiting,—the physician noticed a small ulcer on the left margin of the tongue, one inch from the tip, and cauterized it with silver nitrate. The patient stated that he had first noticed the ulcer two months previously—in September, 1913.

There is a dull ache when eating hot or cold food, but no other pain and no hemorrhage. This ulcer, at first the diameter of a lead-pencil, has gradually enlarged until, at present, it is about the size of a twenty-five-cent piece, is excavated, and has ragged margins. Up until five weeks ago the physician maintained that it was not a cancer, and treated it with the iodids. A piece was snipped off and found, upon examination, to be carcinoma. A

Wassermann reaction, made five weeks previous to admission, was positive.

COMMENTS

Dr. Murphy (June 12, 1914): The patient first noticed the ulcer in September, 1913, which was nine months ago. Two months later, in November, the physician observed a little sore on the left side of the tongue. A little ulcer on the inner side of the tongue is always serious in a man of fifty: if such an ulcer does not heal within a week or ten days without any treatment whatever, except the removal of an irritating tooth or plate, it signifies one of three common lesions. The most common is carcinoma; second, syphilis; third, tuberculosis. Other lesions may occur, but these three are the commonest. Any lesion is of sufficient importance to demand the most serious attention the moment you see it. What do we mean by "*the most serious attention*"? We mean exhausting all available methods of diagnosis—making tuberculin and complement-fixation tests and excising a specimen for microscopic examination. *Do not make an application of silver nitrate or nitric acid or glacial acetic acid!* All these measures do what? They obscure the normal picture of the disease and handicap the next physician who comes in to examine the case.

Now, then, what has happened since last September? This ulcer has advanced from the stage where operation could have been performed with little or no danger to life, to the stage attended by a superlative degree of danger in operation and to a practically certain cancer termination of life. Why? Because the first physician who saw the lesion did not make a positive diagnosis. The time is coming when we shall be punished more severely for sins of omission than for sins of commission, because there is no excuse, no extenuating circumstance for such mismanagement. As it is now we must remove the whole tongue—an operation with a mortality of 40 per cent.—four chances in ten that the patient dies quickly from inhalation pneumonia. This is terrible to contemplate, since the lesion was observed in the early stage, when amenable to cure by a limited operation. In order to realize the situation, just reverse the circumstances—put yourself in the patient's position and see how you would feel!

Upon one side the lymph-nodes are already involved. Now, then, you can see what little chance this patient has for life by considering that epithelioma of the lip has a cancer mortality of 52 per cent. if operated upon before there are palpable lymph-nodes in the submaxillary zone. Think of it! a cancer mortality of 52 per cent.! And when there is palpable enlargement of the lymph-nodes at the time of primary operation, the mortality leaps up to 76 per cent., and yet cancer of the lip *per se* is practically the least malignant of all cancers.

We reckon this man's chances of recovery with permanent cure at about one in twenty—about 5 per cent. But why, you ask, do we perform the operation? Our answer is, that with the exception of cancer of the urinary bladder, death from cancer of the tongue is the most painful of all cancer deaths, especially in the later stages, and it is for this reason that we excise this tongue, not in expectation of curing the disease, but with the hope that by removing the primary focus, even though metastases have already taken place, he might live an easier life and secure euthanasia.

Lesions of the tongue are not at all difficult to differentiate or diagnose. Within twenty minutes after the patient consulted us we had excised a specimen and established the diagnosis. Just cut the specimen with the freezing microtome, mount it upon a slide, and make the diagnosis with the microscope. When one can do that, there is no excuse for delay.

Remember, this is a Chicago patient: he did not come from the country. That fact verifies what we maintain—that the cross-roads doctor is a better diagnostician than the side-street or boulevard doctor. By the latter we refer to the class of physicians in general practice—in practice of every type. In the city the doctors do not get at the case with the positiveness and force which the men out in the country exercise. The cross-roads doctor is less distracted, and seems to be able to employ more horse-sense in diagnosis than the average city doctor can—there is no question about it. He has more time to live, and in the country much less of a struggle for existence. In Chicago, five or six years ago, Dr. Evans made an investigation of the average income of the city doctors, aside from the specialists, and found it to be nine hundred odd dollars a year: this conveys some idea of the struggle and competition in a large city.

This patient is a fairly well-preserved man of only fifty-one years: it just makes me creep to think of what I have to do.

OPERATION

Plan.—Approach through neck and excision of tongue through floor of mouth. Ligation of lingual arteries. Wide excision of all involved tissue to prevent prompt and immediate return.

Findings.—Involvement of submaxillary lymph-nodes, the largest being that in the anterior pole of the submaxillary salivary gland. Tongue involved on the left nearly as far back as the circumvallate papillæ, and a little farther on the right.

Steps.—Removal of submaxillary lymph-nodes, peeling them with the periosteum; removal of sublingual and submaxillary salivary glands; mops with iodin exposed field and floor of mouth before opening into mouth; uses digastric muscle as a guide; lingual artery exposed and ligated; tongue amputated as far back as the circumvallate papillæ, and a little beyond on the right; stump of tongue saved for functions of articulation and deglutition; muscles of floor of mouth apposed and latter closed by suturing mucosa; fenestrated drain inserted; wound closed; specimen sent to pathologist.

Points Emphasized.—In excising the tongue be prepared to work very rapidly; use digastric muscle as a guide; clean floor of mouth to minimize infection of cellular tissue of neck; keep blood from trachea for fear of inhalation-pneumonia; cancer does not travel through musculature of tongue, but through lymph-spaces along midline; preserve stump of tongue if possible.

The patient will be given one-half strength nutrient enemata, but nothing by mouth for several days.

[*N. B.:* The wound healed by primary union.]

[A case of carcinoma of the tongue, together with a talk on the differential diagnosis, was published in the CLINICS for October, 1913, pp. 879–883. A table of tumors of the tongue is given in the same number, p. 872, in connection with a case of tuberculoma of the tongue.]

CICATRICIAL CONTRACTURE OF NECK FOLLOWING A BURN—RESECTION OF SCAR AND INTERPOSITION OF A FLAP OF NORMAL SKIN

Summary: Clinical history of patient; two varieties of scars and positions they occupy; scar cosmetically disturbing to patient to extent of becoming an obsession; treatment by resection and interposition of normal skin; prevention of contracted anus after operation for hemorrhoids; scar following excision of tuberculous lymph-nodes of neck becoming rarer; citation of case of keloid formation in a white woman nursed by a negress; operation.

HISTORY

The patient, a female aged nineteen years, was admitted to hospital July 14, 1914, and recited the following history:

In 1901, when six years of age, while playing around an open grate fire, a tissue-paper bow around her neck caught fire and was consumed in a flash. It burned her neck from ear to ear for a width of 2½ inches. The lesion was dressed and healed within four weeks. At first the skin was smooth, but at the end of two weeks it began to contract, leaving a scar which caused neither pain nor dysphagia.

Eight years ago, in 1906, she was operated upon by the late Dr. Nicholas Senn. An incision was made from ear to ear, the skin reflected, the scar-tissue excised, and the wound closed. A gypsum case was worn two weeks, and after its removal the scar stretched on the right side of the median line.

COMMENTS

Dr. Murphy (July 16, 1914): That is the typical contraction-scar. You have so often heard me speak of the annular scar and the contraction-scar—the two varieties in which there is excess of development of connective tissue even to keloid formation, but, above all, in which there is contraction. The positions these vicious scars occupy are:

1. The flexure of the elbow.
2. The flexure of the popliteal space.
3. The flexure of the wrist.
4. The neck.
5. The leg—in the annular scar following the Schede operation for varicose veins: this procedure is now obsolete, owing to the resulting circular constricting scar.
6. The anus—in the annular scar following the Whitehead operation for hemorrhoids.

Such contracting keloid scars develop in skin that is subjected to alternating forcible tension and relaxation: that is the reason Dr. Senn removed the original scar.

While from my viewpoint that is not much of a scar, yet in her estimation it is a "fierce" scar. She fancies that all passers-by in the street gaze at it, while the truth is that nobody notices it. It has just become an obsession with her.

These disfiguring scars develop in still another situation—after the operation for webbed fingers. The scar upon the extensor surface causes no trouble because it is a little back of the midline. That on the volar surface, however, soon contracts, gradually flexing the finger until you just think it will never stop.

We have two of these cases at present. You recall the scar in the case of talipes equinovarus that we operated upon last Friday. A dissection had been made upon the border of the foot, just at the junction between the dorsal and the plantar tissues. That scar commenced contracting and continued contracting. It was a definite example of the behavior of plantar and palmar longitudinal scars in this class of cases.

We recall the first two cases of the Schede operation in which undue contraction resulted from the complete annular division of the skin around the leg, including the veins. Immediately after the operation the circulation in the foot was good, and everything went along all right. Slowly but surely, however, that scar began to contract. In one case gangrene developed and involved half of the foot, while in the other only one toe was involved at the time of prophylactic operation.

What is the proper method of dealing with these scars? Excise a portion and insert a flap of normal tissue so that between the two ends of the scar there is elastic tissue, that is, normal skin. This yields to the tension: it stretches and stretches.

In anticipation of the contracted anus that follows the Whitehead operation for hemorrhoids, always preserve the skin-tabs on the outside. When contraction occurs, split the skin longitudinally and suture it to the mucosa for an extent of from one-half to three-fourths of an inch. After interposing three such tabs absorption of the scar takes place, because normal elastic tissue has been inserted between the two ends of the connective-tissue ridge.

Occasionally you see scars in the neck after operations of excision of tuberculous lymph-nodes: we do not now encounter them so often because with us the procedure is obsolete—I think it is now eight years since we performed the last dissection of the neck for this disease. We now treat them with tuberculin and sometimes drain the individual mixed infected glands.

At times the scar-tissue develops into keloid. The white race is not subject to the severe types of keloid formation that the colored race is. The source of nourishment seems to play some rôle in these cases. I recall a white patient from Little Rock, Arkansas, who had a mass in the neck as large as a guinea-hen's egg. There was a little group of keloids on the breast also, and another scar upon the opposite arm, half as large as a harvest apple. I had never seen such scars in a white woman. I inquired who had nursed her, and she replied a colored "mammy." That cleared up the situation. She had been nursed from birth by a negress and there was some relationship between the nursing and the development of a keloid typical in blacks, although no negro blood coursed in her own veins.

In this patient there is a typically contracting scar, though not a vicious one. Being a transverse scar it is not subjected to tension upon motion of the neck as it would be were it longitudinal.

OPERATION

We divide the scar-tissue and then dissect up the cicatricial portion of the annular flap, freeing it half an inch at its lower angle and three-fourths inch at its upper angle, in order to overlap the scar. We next insert this graft of normal skin to act as an elastic break in the continuity of the scar: by stretching, it will become two-thirds larger. *That* is the key to success!

In dissecting out the scar the incision should be clean-cut and at right angles to the derma for its *entire* depth: the skin incision should never be beveled, as this tends to keloid or fibrous deposit, makes an unsightly scar, and renders it very hard to cut the skin of the neck at right angles. This is most easily accomplished by holding the skin under tension while the blade is penetrating it. It is equally important on the face and neck that the skin be *accurately* adjusted edge to edge for its entire depth.

It is a little surprising that a tissue-paper burn should be severe enough to have involved the derma, for a burn must extend to the derma to result in contraction.

[A case of "Contracted Cicatrices on Index-finger and Thumb" was published in the CLINICS for February, 1915, pp. 155–163.]

RECURRENT LUXATION OF HUMERUS—CAPSULORRHAPHY

Summary: Clinical history of patient; comments—contrast between small size of glenoid cavity and large size of humeral head; operation; original description of operation; points emphasized.

HISTORY

THE patient, a female aged fifty-five years, was admitted to hospital February 4, 1915. The family and past histories are negative.

The right shoulder has been luxated ten times.

The first time was thirty-one years ago, in November, 1884. When jumping from a wagon, her skirt caught in a nail, flinging her headlong. She landed upon the right elbow, luxating the right shoulder upward and forward; severe pain ensued. She was chloroformed and the luxation was reduced.

The second time was thirteen years ago, in the winter of 1902. While getting into a buggy, with the right hand grasping the seat and the right foot upon the step, the horse started, throwing her backward with her hand still grasping the seat, again resulting in luxation of the right shoulder.

The third time was in the summer of the same year, 1902; the luxation occurred while she was giving her outstretched right arm to her sister for assistance in reaching a step three feet from the ground. The weight of her body pulled upon the arm, luxating it.

The fourth time was in the fall of the same year, 1902, when she slipped on the ice, striking upon the right elbow, luxating the arm.

The fifth time she does not remember the date of, but a luxation occurred while swinging the right arm upward and backward to throw something.

The sixth time was five years ago—in January, 1910. Her chair slid from under her on a freshly polished floor. She does not remember the mechanism, but upon arising the shoulder was luxated.

The seventh time was in the summer of the same year, 1910. This time she was firing china in a kiln, and in stooping over to look under it while at the same time swinging the arm backward for support, the luxation occurred.

The eighth time was sixteen months ago, in October, 1913, and the luxation resulted from slipping upon snow.

The ninth time was eight months ago, in June, 1914. In removing fruit-jars from a shelf with the left hand while the right hand supported her against the wall, the luxation occurred.

The tenth time was the day before admission; the luxation occurred while dressing.

COMMENTS

Dr. Murphy (February 6, 1915): That history is typical for a case of recurrent luxation of the shoulder. Contrasting the glenoid cavity with the acetabulum, you see that the latter is a cavity of considerable size, while the glenoid is just about as much a cavity as the large end of a billiard cue is. Compared to the large size of the humeral head, the glenoid is so small as to make one wonder that, when once luxated, the head can be made to stay in contact with the glenoid again.

OPERATION

[Dr. Murphy exposed the capsule by incising the anterior portion of the deltoid muscle; then by retraction he exposed the head and brought into view the bicipital groove, opening the latter to be sure of its identity. With the humerus rotated externally he divided the capsule, freed the anterior half, and, reflecting it externally, united it to the posterior attachment of the humerus (Figs. 204, 205, 206). All the deeper work was performed without hand-contacting.]

Dr. Murphy: In our original description of the operation we elevated for one inch the portion of the capsule that covered

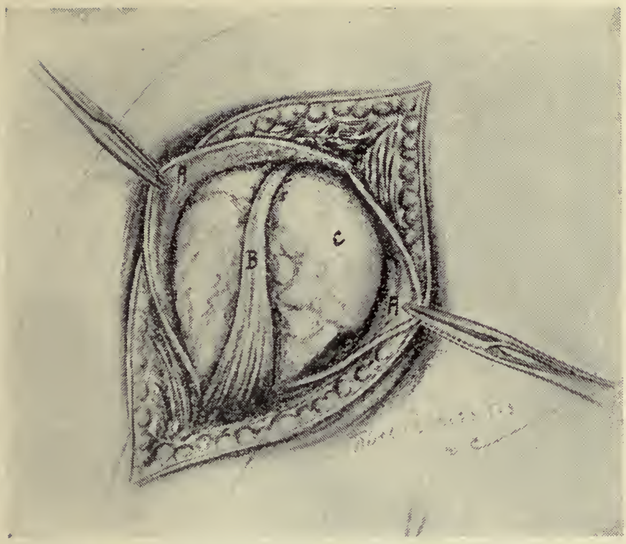


Fig. 204.—Murphy's operation for recurring luxation of the shoulder-joint. Incision anterior to bicipital groove, through deltoid muscle, exposing the joint. A, Capsule split open; B, biceps tendon in bicipital groove; C, head of humerus.

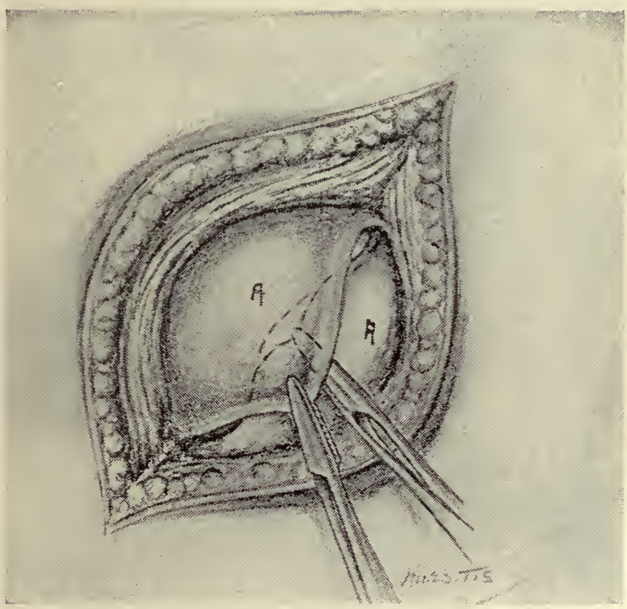


Fig. 205.—Overlapping or imbrication of capsule so as to reduce capsular tissue to desired amount, thus increasing retentive power, so as to prevent luxation of humerus.

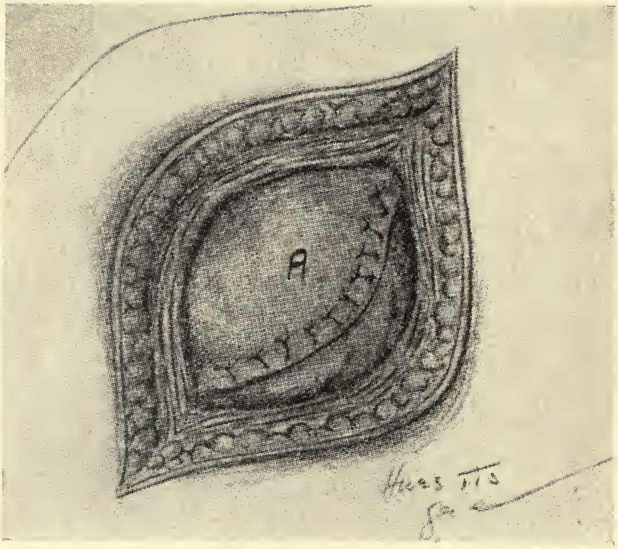


Fig. 206.—Capsule sutured, to be followed by usual closure of wound.

the long tendon of the biceps muscle and, dividing it behind, made an edge-to-edge union of the capsule. Such union affords the greatest security.

[A description of this operation was published in *Jour. A. M. A.*, vol. lxviii, 1912.]

Points Emphasized.—Incise the deltoid muscle as far forward as possible in order to minimize the damage to the circumflex nerve, paralyzing the least number of muscular fibers.

Hand-contacting in bone and joint work is dangerous. If you can keep your hands from the wound you may be fairly sure of obtaining an aseptic result.

[Primary union resulted. When last heard from there had been no recurrence.]

SUBCORACOID LUXATION OF HEAD AND FRACTURE OF SURGICAL NECK OF HUMERUS—OPERATIVE REDUCTION MAINTAINED BY PLATING

Summary: Clinical history of patient; comments—unreliability of skiagrams in shoulder injuries; removal of head of humerus or of femur and survival if replaced; operation deferred until tissues have been coffer-dammed; operation—no-hand-contacting method essential; finding of luxated head refuted skiagram; steps of operation; remarks upon the Dupuytren contracture.

HISTORY

THE patient, a physician aged forty years, was admitted to hospital December 26, 1914. The family and past histories are negative.

Four days before admission, on December 22, 1914, the patient was thrown out of a buggy, falling and striking the left shoulder against a rock; he was unconscious fifteen minutes. A physician failed to reduce the resultant deformity. The arm was discolored from shoulder to elbow. Skiagram revealed fracture of the surgical neck of the left humerus and subcoracoid luxation of the capital fragment.

Twelve years previously, in 1902, Dupuytren's contracture of both hands developed gradually without definite cause. The right hand has been operated upon. In the left hand the contracture is most marked in the little and ring-fingers.

COMMENTS

DR. MURPHY (January 2, 1915): From the appearance of the skiagram it does not *seem* that the head is absent from the glenoid cavity (Fig. 207). You can see in both the anteroposterior and the lateral views, even with the arm rotated outward to a right angle, that the head still remains in the glenoid caity—*apparently*.

We show a skiagram from another case in which the x-rays failed to reveal the relation of the head of the humerus to the glenoid cavity. When you look at the skiagram you say the humeral head is in perfect apposition. From another angle the skiagram still shows good position, yet physical examination determined the absence of the head from the glenoid cavity and its presence behind and beneath the acromion process of the scapula. *The physical examination belied the skiagram.* Of course, the physical examination was correct because the head could be felt away out by any one with but little knowledge of anatomic relations. Many months after the accident we removed the head and placed it upon the table, exposed the glenoid cavity and replaced the head, and it throve. We have had five similar cases and the results have been good in all.

In but one case was the head of the femur removed onto the table and replanted. A patient fifty-five years of age suffered a fracture of the neck of the femur with coincident luxation of the head. About one and a half years after the accident we removed the head from its luxated position, replaced it in the acetabulum, and nailed it to the neck, securing perfect union and revivification of the head. The basis of this procedure was an operation we performed five years previously. A boy of eleven years sustained a fracture of the neck of the femur in which the head was rotated outward and the round ligament strangulated, shutting off the circulation. At operation, seven weeks after the accident, we found the head dry, bloodless, and black—in effect, necrotic. Nevertheless, we freshened the head, turned it into the acetabulum, and nailed it in place. Notwithstanding that seven weeks had elapsed since the accident, a perfect result was obtained. It is now five years since the operation. In April we demonstrated him before the International Surgical Congress, and at that time he could walk without revealing the slightest clue to the side injured.

This patient's injury was received but four days ago. We never perform these operations immediately after the accident, but wait until the tissues have reached the granulation stage of repair—usually until the twelfth or fifteenth day. At that time

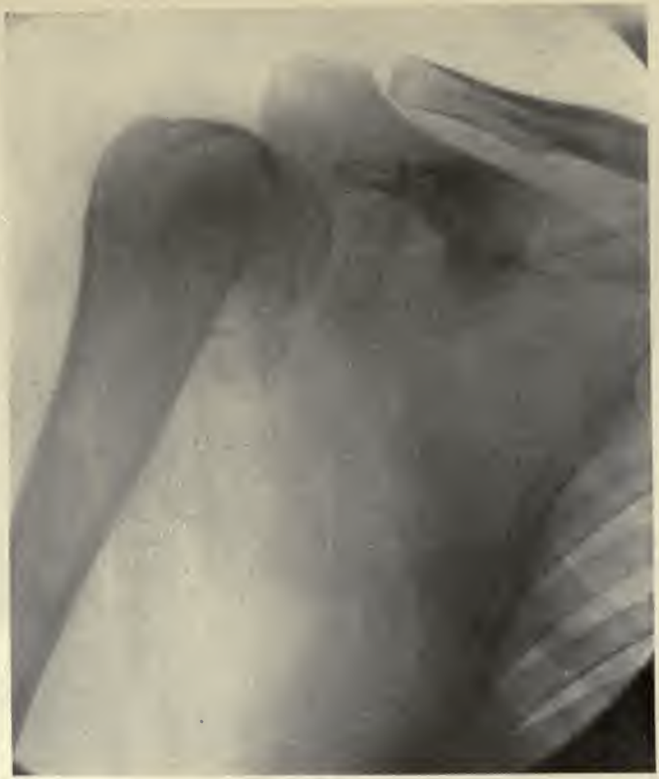


Fig. 207.—Preoperative skiagram, showing fracture-luxation of upper end of humerus; fracture of surgical neck and subcoracoid luxation of head. The luxation is hard to detect in this anteroposterior view. Note that proximal end of distal fragment rests upon head, causing shortening of the humerus.

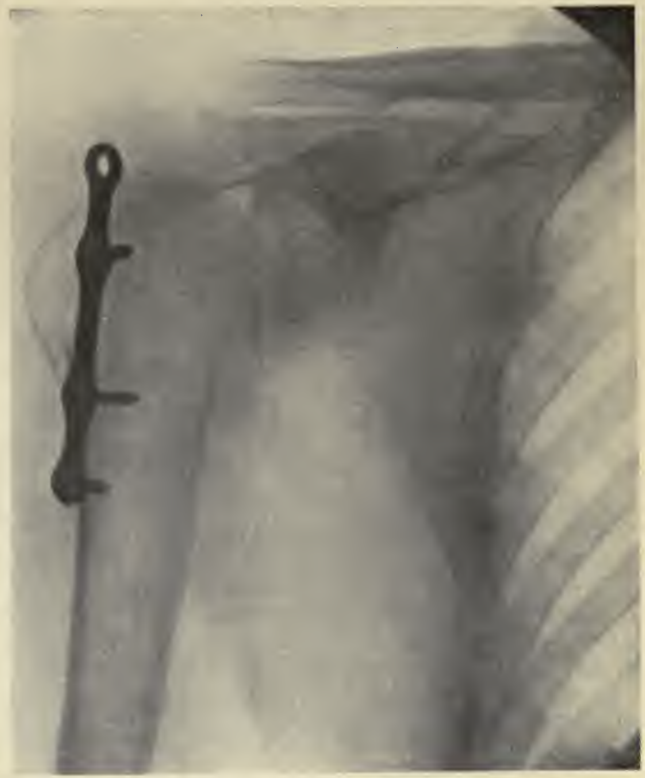


Fig. 208.—Postoperative skiagram, showing reduction of fracture-luxation of upper end of humerus and maintenance of reduction by a plate. Note that but one screw could be applied to the capital fragment (anteroposterior view). Deformity reduced and the shaft in the proper relationship to the head.

the lymph-spaces are coffer-dammed against infection, while before that time the tissues are in the lowest state of resistance.

You must endeavor to do all bone operations without hand-contacting. That is the key to asepsis. In determining the positions of the fragments, substitute the long forceps for the fingers.

Notwithstanding the skiagram, I should say that the head is anterior to the glenoid cavity and *not* in it.

Let the record show that the head rested anterior to the glenoid cavity, and a little below when the arm was up in a straight position, belying the appearance in the skiagram and accentuating the observation we made upon the unreliability of skiagrams of the shoulder.

OPERATION

[After exposing the head through an anterior incision the deformity was overcome by removing a fragment from the comminuted fracture, so that the head and neck were levered into the same parallel plane. Then a Lane plate, in which only one screw could be applied on the proximal side, as the other screw came over the comminuted portion of the head, was inserted to prevent the head from sliding inward, whereupon the conformation was accurately restored (Fig. 208).]

DR. MURPHY: The patient has also Dupuytren's contracture on this side. We shall not operate upon that now because of the slight danger of ascending infection to the shoulder. The contracture of the right hand has previously been operated upon.

[There was primary union.]

[For the report of a case of subcoracoid luxation with avulsion of the greater tuberosity, together with comments on the luxation in general, see CLINICS, June, 1913, pp. 413–420.]

GUNSHOT WOUND OF ARM. CICATRICIAL COMPRESSION OF ULNAR NERVE—RELEASE; DIVISION OF MEDIAN NERVE—NEURORRHAPHY

Summary: Clinical history of patient; comments—remarkable course of bullet through arm; operation—search for ulnar nerve; ulnar nerve compressed and not divided; search for median nerve; median nerve divided; resection and suture of median nerve; nerve wrapped in egg-membrane and line of union surrounded by flap of fascia and fat; correlation of clinical signs with operative findings; mode of destruction and of repair of nerves; time of regeneration of nerves and of muscles; gradual resumption of extension.

HISTORY

The patient, a male aged forty years, was admitted to hospital September 23, 1914. The family history is negative. Past history: When fifteen years of age he had typhoid fever and was sick two months.

Two months ago, on July 28, 1914, the patient, while sitting down with the left arm in his lap, was shot at from behind by a man with a 45-caliber Winchester rifle. The leaden bullet entered the posterior surface of the left arm four inches above the olecranon process, and emerged at the flexure of the elbow near the midline; it then struck the abdomen above the level of the anterior superior spine, passed under the skin, and emerged two inches below the navel and an equal distance from the median line on the same side.

At the time of the shooting the patient sensed excruciating pain at the elbow, which continued, diminishing in severity, for eleven days, until the wound was healed. Just after the affray he could neither extend, nor flex the forearm, nor could he move the fingers. There was numbness from elbow to finger-tips. The wound bled profusely.

Two hours after the accident he was admitted to a hospital.

Shortly afterward the patient noticed that the fingers of the left hand were flexed, and they remained flexed two days. For an equal period there was no sensation in the whole forearm, nor in the hand, upon pricking the skin with a pin. Gradually, however, sensation returned—first in the back of the forearm.

Two hours after the accident the patient was anesthetized at the hospital, and some of the bleeding vessels were ligated. Through-and-through drainage was inserted and maintained three days. The arm was dressed in a slightly flexed position, resting upon a pillow. Dressing was performed every other day. He remained in bed nineteen days.

At the end of the twelfth day after operation the pain left the elbow, but ever since then there has been pain in the whole hand, with the exception of the thumb and index-finger. Numbness now involves the forearm except the dorsum, and the whole hand as well. When the hand is supinated he cannot flex the fingers. If the hand be pronated, he can extend the fingers. He can adduct and incompletely abduct the thumb. There is good motion at the elbow.

COMMENTS

Dr. Murphy: This man was shot in the left arm just above the elbow. I take it that the bullet entered and divided the brachial artery and the median nerve just above the antecubital fossa, judging from the line of the operative incision and from the inability to flex any of the fingers or abduct the thumb. The distribution of the paralysis indicates injury to the ulnar as well as to the median nerve. The remarkable anatomic feature is that the bullet in its course could injure both these nerves and injure them without puncturing the bone!

The missile encountered first the ulnar nerve and then traveled forward and outward along the line that led directly to the median nerve, resulting in immediate loss of power in the hand. We must locate the median and the ulnar nerves. He had a severe hemorrhage, and this field was opened for the purpose of ligating the brachial artery, and the resulting postoperative cicatricial tissue will make it a little more difficult to find the nerves.

OPERATION

I told you this morning that I would have to deal with the ulnar nerve twice: to expose it I shall have to go quite a little higher than was necessary in the other operation, and I shall have to go in from before, although it is easier to reach the nerve from behind.

[Incision.] There is the ulnar nerve, just where we left it at the previous operation. In the position my finger occupies I cannot feel from the outside a bulbous end of the nerve.

This patient should have an exceedingly well-developed ulnar artery, unless the ulnar artery was ligated, which was not the case. It was the *brachial* artery that was ligated, and the branch that accompanies the ulnar nerve—the inferior profunda artery —very frequently takes up, in conjunction with the anastomotica magna and the posterior ulnar recurrent arteries, the collateral circulation when the brachial artery is out of commission.

Now we retract the inner head of the triceps muscle, because the ulnar nerve should be just to the inner side of it, and beneath it. There is your nerve! See it shining in the field! We next determine whether the ulnar nerve is divided or merely compressed or congested. Here is the venous plexus at the bend of the elbow.

We already encounter cicatricial tissue. I am working on the inner side. Above, you see the proximal portion of the nerve, and below, the distal portion. Let us see whether the distal portion is bound down. We test it with the exciter. See! The adductor pollicis contracts, showing that the axons are alive and indicating that the loss of transmission is the result of compression only and not the result of division. This continuity of the nerve explains my failure to feel a neuroma on palpation, for no neuroma was present. As I free the nerve from the scar-tissue that binds it down to the bone you see the flexor carpi ulnaris contract from the irritation, in addition to the adductor pollicis. There is the bullet-track. After lifting the nerve from the scar-tissue we displace it forward to beneath the biceps muscle, and intend to fix it there permanently where it will be free from any future danger of compression.

We had a case very similar to this one in an injury to the musculospiral nerve, which had dwindled down to just a little cord-like mass of tissue at the point of cicatricial compression. When I applied the Faradic test below the point of compression the nerve responded, showing that it was alive.

I know that this ulnar nerve is not degenerated below, for if it were there would be no response. Why? Because the adductor pollicis is one of the last muscles to be supplied by it. The ulnar nerve supplies all the muscles in the palm of the hand except the abductor pollicis, the opponens pollicis, the outer head of the flexor brevis pollicis, and the outer two lumbrical muscles. There is the track of the bullet: it shows as a white line where the missile perforated the muscles. What was the date of this injury?

INTERN: It occurred eight weeks ago.

DR. MURPHY: One of the doctors here has just cited a case in which a man was injured just above the elbow by the horn of a charging cow. There was a strip of tissue hanging out of the wound, and the doctor pulled it out farther and cut it off. Immediately there was loss of extension of the wrist! What had happened? The doctor had cut the musculospiral nerve.

Let the record show that the ulnar nerve was exposed, found compressed by cicatricial tissue and freed, and that before exposing it we could not palpate any neuroma.

Now we search for the median nerve, and one of the things to which we wish to call your attention is the presence of that cicatricial mass at the flexure of the elbow. Wherever you have a vertical incision crossing the flexure of a joint you always have a disagreeable situation to deal with. Here in the case of the elbow the median nerve folds in as the scar contracts. We would like to feel a neuroma, but we cannot palpate any; we can feel only a thickened mass. Remember, ligation of the brachial artery was performed here opposite the flexure of the elbow, and we have the operative scar to contend with, so that we must go higher up along the inner side of the biceps muscle through the scar-tissue to locate the median nerve. We divide the median basilic vein.

It is very helpful in searching for this nerve to have a pulsating brachial artery in the field as a guide. In this case the brachial artery has been ligated. Not only do we not have it in the field, but we shall be bothered by the strand of connective tissue that has formed along the path of the artery. That is the annoying feature of this mass of connective tissue.

I am going above and to the inner side of the belly of the biceps. Now I am in the midst of a cicatricial mass where I cannot recognize a single anatomic structure. That structure I take to be the inner margin of the biceps muscle. There is the median nerve shining in the field! Now we are approaching the neuroma. There is the bicipital or semilunar fascia; the nerve is always beneath it. The fascia is one of the best guides to the position of the median nerve, and shows up beautifully here as it runs from the tendon of the biceps downward and inward across the ulnar border of the forearm. Just below the crossing of the bicipital fascia the median nerve undergoes subdivision. Now we are orientated in both the upper and the lower portions of the wound.

Let the record show that there was a marked contrast in color between the portion of the nerve above the wound and that below; that the nerve is white above and pinkish below, showing the changes that are associated with fragmentation of the axons and degeneration in the case of the lower portion.

Let the record show further that the perineural tissues are edematous, signifying the reaction to regeneration that one would see around an infected nerve.

Now I am dividing the connective tissue.

Let the record show that the scar-tissue is much denser in front than behind, and that there is a neuroma in the middle of the nerve.

There apparently is the track where the bullet went through the nerve. In its course the bullet appears to have split the median nerve. Let us investigate the condition of the axons of the distal end below the bullet track. We take off the constrictor. I want to see how many of these axons are still living, to determine whether or not I shall resect the nerve. The nerve is entirely out of commission, since it gives no response to the Faradic current. Now we must make a resection of the nerve and reunite it end to end, after freeing it well above and below the zone resected. I shall stop all these bleeding points in preparation for the resection.

The next step is to divide the nerve. You can see the path of the bullet as it penetrated the nerve from within outward. See the fasciculi, or the brush-ends of the nerve, standing out! They indicate that we have at last reached a normal portion of the nerve. The best guide as to whether we are above and below the site of the neuroma, *i. e.*, in normal nerve-tissue for union, is these little brush-like nerve-ends when you see them sticking up, for then you know that you have prepared the nerve far enough back from its traumatized portion for regeneration to take place.

I take in the bite of my needle the perineural connective tissue on one end of the nerve, and again the same tissue at the corresponding point on the other end, in an endeavor to restore the former axonal relationship as nearly as possible. It does not appear to matter materially whether we connect the same axons again, provided we get an axonal contacting. I am using for suture material chromicized catgut.

What we need above all things is to prevent the interposition between the nerve-ends of ordinary connective tissue from the sides. Remember, these nerve-ends were united after the injury not by nerve-tissue, but by a coil of connective tissue, which is merely the normal histologic structure that pads the spaces between muscles, blood-vessels, and nerves. Now I have the nerve united by three stitches, and these will suffice to hold it in position. Using egg-membrane we next wrap the nerve merely to prevent the interposition of common connective tissue from the side. In preparing this egg-membrane the egg is just boiled hard and the membrane removed. You wrap the joint in the same way that the electrician does. In fixing the egg-membrane in place we insert one stitch above and another below, so that it cannot get away unless we exercise undue traction upon it.

VISITING DOCTOR: Doctor, in the final result will there be any harm done by lengthening that nerve when the arm is straightened out?

Dr. Murphy: No. The nerve will in time be strong enough to permit of stretching out without giving way, but in the beginning after operation we must keep the arm flexed for a great many weeks. Now I have prepared a pedicled flap of fascia and fat to encircle the line of union and prevent a mass of connective tissue from working in upon it and constricting it.

Let the record show that in addition to the egg-membrane we placed a capsule of fascia and fat around the line of union of the nerve so as to prevent the cicatricial tissue from interfering with the transmission of impulses upon the completion of repair.

Let the record show further that the arm was dressed in the position of flexion to prevent tension upon the line of union of the median nerve; that we resected half an inch of the median nerve before reaching sound tissue, as shown by the brush-like ends appearing; that the bullet had passed through the nerve at an angle; that when we made the Faradic test there was not the slightest contraction of any muscles supplied by the median nerve, but that, on the other hand, when we exposed and freed the ulnar nerve and applied the Faradic current to it below the site of compression we were able to elicit immediate contraction of the muscles supplied by the ulnar nerve.

RECAPITULATION

Now we shall summarize the case. The nature of the injury that involved the ulnar nerve was a cicatricial compression, and this scar-tissue caused suspension of transmission of impulses through that nerve. When we exposed the nerve and freed it from the cicatricial fixation the axons were still alive, because it is only a living axon that will give a response to the Faradic current.

Contrast the *compression* of the ulnar nerve in this case with the case we had on Monday, in which the ulnar nerve had been *divided*. In the case of the divided nerve, after exposing the line of division and applying the Faradic current to the distal end, we got a positive response, as was shown by contraction of the muscles supplied by the nerve. The reason that the distal end responded positively on Monday was that we applied the Faradic current before axonal degeneration had had time to take place, since transmission of impulses continues as long as eighteen hours after division of a nerve. If we should apply the Faradic current in the case in which the nerve had been divided after the lapse of a week, there would not be the slightest response—just as in the divided median nerve of this patient, now two months after the accident, there is no response owing to degeneration of all the distal axons.

We made an apposition of the median nerve mechanically. Mere apposition of the nerve is not the important factor, because you can take nerves that have been completely divided, approximate them with the scar-covered bulb-ends still on the nerve, and put them in good apposition, but there will be no return of transmission until the bulb-ends are removed. What you must do—and this is the important factor in reuniting nerves—is to make an axonal contacting without the interposition of any foreign body or of any connective tissue.

It is now two months since this man was shot in the arm. What has been taking place in the distal portion of the nerve during that time? The first thing that occurred was the rapid degeneration of the axis-cylinder processes, and after that the degeneration of the myelin-sheath. Finally, the fibers of the muscles supplied by the nerve shriveled up, but not beyond the capability of regeneration.

Now let us take up the stages of regeneration of the nerve after operation. The first stage is that of congestion of the cells that compose the neurilemma. Probably after that stage of congestion there is reconstructed within the neurilemma a new embryonal myelin-sheath and axis-cylinder. Kennedy believes that there is reconstructed a new embryonal tissue resembling the axis-cylinder process before the nerve is sutured, but that it is not capable of transmission. This hypothesis we question. After the new myelin-sheath is formed, and as soon as the continuity of the nerve-sheath is reëstablished, the tubes for the axons, which tubes are already reconstructed, will be filled up and occupied by the axis-cylinders growing down from the proximal axis-cylinders, very much as rising mercury in a thermometer. These axis-cylinders will run right down through the tubes as the tubes are formed by the neurilemma, and they will finally travel down to the most distal portion of the nerve.

In the case of the axis-cylinders this regeneration proceeds entirely from the proximal side and does not take place from the distal side, for the axis-cylinder is merely an elongation of the protoplasm of the parent nerve-cell body that resides in the spinal cord. The neurilemma cells, however, unlike the axis-cylinders, regenerate the sheath of Schwann all along the distal side, because the neurilemma consists of a fine, homogeneous, elastic membrane with a neurilemma cell at each internodal segment. What results if you cut off any portion of a cell from its nucleus? The death of the distal or excluded portion of the cell, since it can obtain life or regenerate only from the portion containing the nucleus, and therefore regenerates only in a centrifugal direction. The cut axis-cylinder is but a separated portion of the protoplasm of the cell.

How long will regeneration take? If there is no interposition of connective tissue, in from ten to twelve weeks there will be some return of motion. In proportion to the amount of connective tissue that is interposed between the nerve-ends regeneration will take longer, so that in from six or seven months to a year after operation there is practically full restoration of transmission and some return of motion, but not a full restoration of function of the muscle-cells.

What effect has the division of a nerve upon the muscle-cells? First the muscle-cells atrophy, but they never degenerate. For perfect restoration of motion there must be a restoration of the muscle-cells. This restoration occurs as soon as the muscles are put to active use. Restoration of the muscles takes longer than regeneration of axis-cylinders. It will be from ten to twelve months before this patient has a good grip in his hand.

The forearm must be kept in the position of flexion for a long time. Then we shall slowly bring it down in gradually increasing ranges of extension. The nerve will have reunited by that time. There is one factor that will limit the degree of extension—it is that scar in the flexure of the elbow. There will be some advantage in this scar, however, in that it will prevent the patient extending the forearm either too rapidly or too far.

FRACTURE OF HUMERUS, LOWER END—FRACTURE OF ULNA, OLECRANON PROCESS—LACERATION OF ULNAR NERVE. OPERATIVE REDUCTION OF FRACTURE—ARTHROPLASTY OF ELBOW-JOINT

Summary: Clinical history of patient; comments and operation; amount of scar-tissue makes arthroplasty difficult; cause of enormous bone proliferation; release of ulnar nerve from scar-tissue and tests for its identification; exposure of bony landmarks about elbow-joint; excision of block of bone that interferes with flexion; motion of elbow-joint restored; interposition of flap; recapitulation; postoperative treatment.

HISTORY

The patient, a male aged twenty years, was admitted to hospital August 26, 1914. The family and past histories were negative.

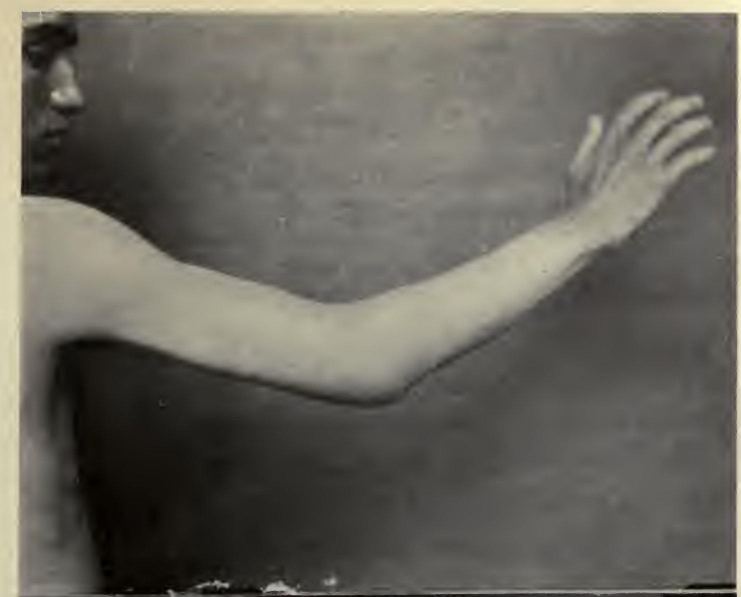
The present injury was received several months ago. While running the patient tripped and fell upon a wheelbarrow. The whole weight of the body came down upon the right hand, which was spread open. While at the time of the fall he did not notice any pain, a few minutes later the whole right forearm and hand were numb, and a few hours after the accident he felt a distinct pain in the hand and a slight ache in the elbow. On getting up from the wheelbarrow the arm was slightly flexed and the elbow seemed to be broader at its tip than it was before the fall. A physician straightened the arm and applied a light gypsum case. At the end of six weeks the case was removed and passive motion instituted. Upon removal of the gypsum case the elbow was stiff, and any attempt at motion caused severe pain. A few days later the arm was bent to almost a right angle. The lost sensation in the hand gradually returned. He is now able to pronate and supinate the forearm and to flex it to a right angle with the upper arm.

COMMENTS

Dr. Murphy (September 9, 1914): Here is a feature I want to call your attention to, namely, that there is a fracture and a displacement backward of the lower fragment (bearing the forearm) which caused stripping up of the periosteum from the humerus for a distance of four inches (Fig. 211). You see the enormous production of callus around the fragments. This callus is worse to deal with than a complete bony ankylosis of origin other than that of trauma. You would think that so long as the lower articular surface of the humerus is preserved intact you would be able to get motion by the interposition of a flap of fascia and fat. If you perform arthroplasty on that basis alone you will have a stiffer joint after you get through with the operation than you have now. You have to deal with this problem entirely on the basis of a bone transplant.

This is a beastly case to work with. The tissues around the joint are infiltrated to such an extent that you cannot fashion an interposing flap of fascia and fat. The arm has no fat remaining on the deep fascia with which to prepare an interposing flap. There is evidence here of a constriction of the nerve by the cicatricial tissue. That is the difficult feature in this case. I formerly had the nightmare when I had one of these cases to deal with, but now it is easier to operate upon them than it used to be to go to Sunday-school.

This excessive production of bone does not look like a traumatic ossifying myositis: I think it is a proliferation consequent upon the elevation of the periosteum. The bony mass is situated upon the postero-internal surface of the humerus (Fig. 212). It is upon the anterior surface of the humerus that the muscle, the brachialis, is attached closely to the bone. The situation here, with the excessive callus, practically amounts to a bony ankylosis. The flexion contracture of the fingers looks as though he has had compression myositis, notwithstanding the fact that he has now recovered fair use of the hand and fingers.



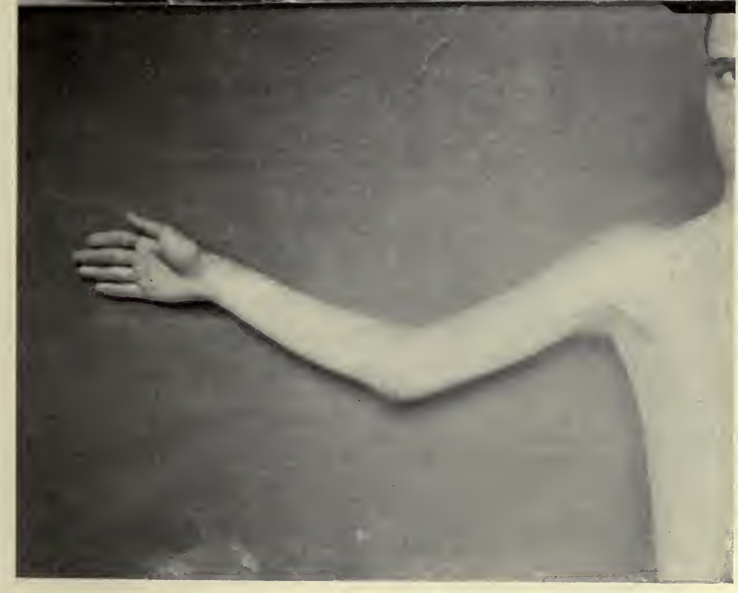


Fig. 209.—Fracture of elbow showing deformity previous to operation. Note bulging just above olecranon process. Note also the slight degree of flexion-contracture of the fingers (external view).

Fig. 210.—Fracture of elbow showing deformity previous to operation (internal view).

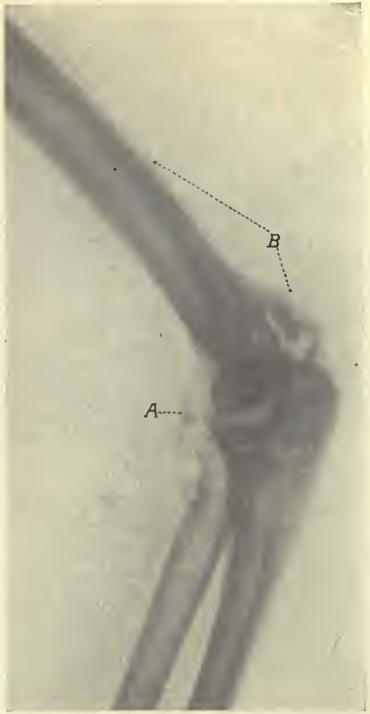


Fig. 211.—Preoperative skiagram showing fracture of lower end of humerus and of olecranon process of ulna, with vicious exostoses and excessive callus formation. *A*, Shows the exostosis extending from the tip of the coronoid process over the anterior surface of the humerus. *B*, Shows the exostosis on the posterior surface of the humerus extending upward for a distance of three or four inches (lateral view).

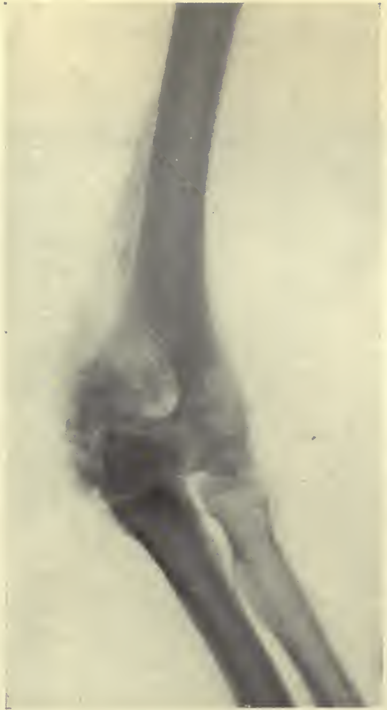


Fig. 212.—Preoperative skiagram of fracture of lower end of humerus and of olecranon process of ulna. In addition to the deformity there is seen excessive subperiosteal bone-proliferation, which begins below, at the internal condyle, and extends upward in relation with the internal supracondylar ridge for a distance of three or four inches (antero-posterior view).

OPERATION

[Incision and exposure of bones.]

There is the olecranon process of the ulna, and here the internal epicondyle of the humerus. The ulnar nerve should rest right in this groove between the olecranon process and the internal epicondyle. I wish to expose the ulnar nerve first so as to put it out of harm's way. There is the ulnar nerve, and one can be sure of its identity by pinching it with the forceps and watching the muscles supplied by it contract; but the use of forceps for the test is not quite so satisfactory as the use of the Faradic exciter. Now I must try to release the ulnar nerve from the mass of cicatricial tissue that has formed as a sequence of the fracture. We are separating it entirely from its capsule of fibrous tissue.

Let the record show that at the site of the injury there is a small neuroma on the ulnar nerve, the result of the trauma, showing that some of the connective tissue within the nerve has been injured, and probably some of the nerve-fibers, or axons, as well.

Now you see the ulnar nerve again, distal to the neuroma, where it passes between the two heads of the flexor carpi ulnaris muscle anteriorly. There are the fine articular filaments which supply the elbow-joint with sensation, and we must divide them in freeing the ulnar nerve. I shall work down upon the bed of the nerve, free it from the connective tissue, elevate it, and throw it entirely out of the field.

The next step is to expose the external condyle of the humerus and remove from it the superficial layer of periosteum, leaving the deep layer attached to the bone. This bone appears to be the lower portion of the internal condyle. There you see the olecranon process, so it is evident that the humerus is injured a considerable distance anterior to the site indicated by the shadow cast in the skiagram. It looks as though there were an elongation of the internal condyle which extends downward and inward from the site of fracture, and which is not shown in the skiagram.

There is the highest point of the internal condyle, and here is the lowest portion of the tip of the elongated fragment. This elongated process will have to come off in order to get a restoration of motion. See! The coronoid process cannot come up to the coronoid fossa of the humerus because it strikes the elongated bony process of the internal condyle. Each case of this type must be treated individually. You can all see the line of fracture. I have already freed the ulnar nerve so that now I can throw it entirely out of the field.

Now we are resecting the internal condyle of the humerus. The next step is to take out the mass of bone that is situated in front. I am not taking off the periosteum, but am leaving it attached to the process of bone to be removed with the condyle. Instead, I am dividing all the tissues in front of the bone and superficial to the periosteum and displacing them out of the operative field so as to gain access to that block of bone and remove it. I am now taking out the bony process with its periosteum without dividing the vessels or the nerves that are situated in front of it. Now you will see just where the elongated internal condyle strikes against the coronoid process. I am now taking out the callus that is behind the joint. The most difficult part, as you will note, to detach is the internal condyle because of the muscular attachments that are still firmly fixed to it; but the condyle must come off together with its periosteum—otherwise the result will be unsatisfactory.

We find here, running up from the lower surface of the humerus and behind the olecranon fossa, a mass of bone. We must remove this mass with its periosteum, so as to let the olecranon process come forward into apposition with the olecranon fossa. Now you see the olecranon process can travel through the full range. The ulnar nerve has been out of the field and therefore out of danger of injury all the time we have been working. The whole secret of success is to prevent a refixation after operation. A little more bone of that callus must be removed. At last you see there is established a full range of flexion and of extension of the elbow-joint.

The next step in the operation is to obtain an interposing flap, and it is a very difficult proposition. I want to try to get all the tissue for a flap that I can. Now I have prepared from the forearm and shall interpose the pedicled flap. I am suturing the remains of the capsule into position. You can see the flap interposed between the bones. I can readily spread the flap by inserting a single stitch. I have now covered the full width of the bones with the flap. There is the base of the internal condyle, and here the group of muscles still remaining attached to the internal epicondyle. We are just refixing them into place.

That operation was slow and tedious, and the hardest arthroplasty that we have ever had to perform.

RECAPITULATION

Let the record show that there was a fracture of the lower end of the humerus with a forward displacement of the distal fragment and a fracture of the olecranon process of the ulna with a backward displacement of the tip of the process; that an extensive exostosis involving the articular surface of the lower end of the humerus in the neighborhood of the internal condyle was excised, and the conformation of the elbow-joint restored so far as the anterior and inner surfaces of the condyle were concerned; that the next step was the implantation of a pedicled flap from the forearm between the ends of the bone to prevent subsequent union; that preceding the operation the range of motion of the elbow-joint did not exceed one inch; that after the joint had been exposed it was found that the tip of the coronoid process impinged against the displaced lower end of the humerus so that it rendered flexion impossible; and, finally, that at the completion of the operation we were able to flex the forearm fully upon the arm.

Postoperative Treatment.—This patient will have no passive motion for about five weeks. After the expiration of that time we shall begin passive motion. The forearm will be dressed in a wire-gauze splint at right angles with the upper arm.

[*Note.*—There was primary union. A good functional result was secured.]

UNUNITED FRACTURE OF INTERNAL CONDYLE OF HUMERUS—REDUCTION OF DISPLACED FRAGMENT AND RETENTION BY EXTRA-ARTICULAR DRILLING

Summary: Clinical history of patient; comments—skiagram misleading; importance of clinical examination in diagnosis of fractures; compact tissue at lower extremity of humerus a site of non-union; replacement and retention of internal condyle by a drill; drill should not be removed to substitute a nail; the extra-articular operation; dangers of bicondylar fractures of humerus; tight bandaging without reduction a fallacy and a cause of Volkmann's contracture; tenoplasty for ischemic contracture; early hyperflexion of elbow another cause of contracture; forward displacement less serious than backward; lower fragment of humerus moves with forearm in non-union; importance of no hand-contacting in bone work; recapitulation.

HISTORY

The patient, a female aged thirty years, was admitted to hospital January 13, 1915.

Nearly four weeks ago, on December 18, 1914, while walking along the street with hands in her muff, the patient slipped on the icy pavement and fell, striking upon the left elbow; the injury was so prostrating that she was unable to get up unassisted. After the accident the arm was very painful and began to swell; she does not remember whether ecchymosis was present. A physician applied a right-angled splint to the arm. A skiagram revealed fracture of the internal condyle of the humerus with displacement of the condyle upward and backward (Fig. 213). Acting upon the skiagraphic findings, the patient was anesthetized and an attempt made to reduce the fragment. After this attempt at reduction the limb was fixed in the position of extension by a gypsum case. At the end of five days the case was removed but the fracture was found to be still unreduced. The patient was anesthetized again and a second attempt at reduction made.

This time the arm was secured by a Velpeau bandage. Since the injury she has had severe pain both day and night.

Examination of the arm shows that the olecranon bears the proper relation to the internal condyle, but not to the external, which is partially obscured by swelling due to traumatic synovitis. The upward displacement of the internal condyle gives rise to great deformity.

COMMENTS

Dr. Murphy (January 20, 1915): Here is a very beautiful illustration of how misleading a skiagram may be. You will note when you look at this skiagram, which was taken in the lateral plane, that there is apparently but little displacement. The line of fracture is almost straight. The internal condyle was displaced upward and backward and the physician dressed the arm in the position of flexion to about this degree [indicating]. Nothing was wrong with the position of dressing, which was the best for a fracture at the elbow-joint; but you see that reduction could not be made by position of dressing alone. You see why this elbow appears normal in the lateral view in the skiagram. The real situation of affairs is masked, showing how important it is to have skiagrams taken from different angles.

In attempts to show posterior luxation of the head of the humerus we have made as many as seven skiagrams, and even then the luxation was not revealed. By clinical examination we could locate the displaced head: it was behind the glenoid cavity and even below the spine of the scapula, but we could not see the displacement through the skiagram. In my teaching to the student body I use that illustrative case to accentuate the fact that, after all, the skiagram forms but one element in making a diagnosis. This unreliability of skiagrams indicates that, in addition to having skiagrams taken, you must cerebrate in order to arrive at the correct conclusion. Even though you might err then, yet you will err less frequently.

These elbow injuries are unpleasant ones to handle primarily, and still less pleasant when you meet them a month after the accident. In this case, at the end of one month, there is no

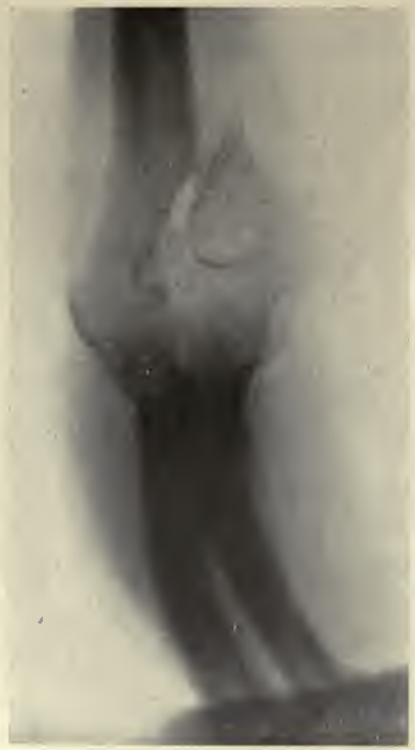


Fig. 213.—Preoperative skiagram of ununited fracture of internal condyle of humerus. Note extent of separation and of upward displacement of the condyle (anteroposterior view).

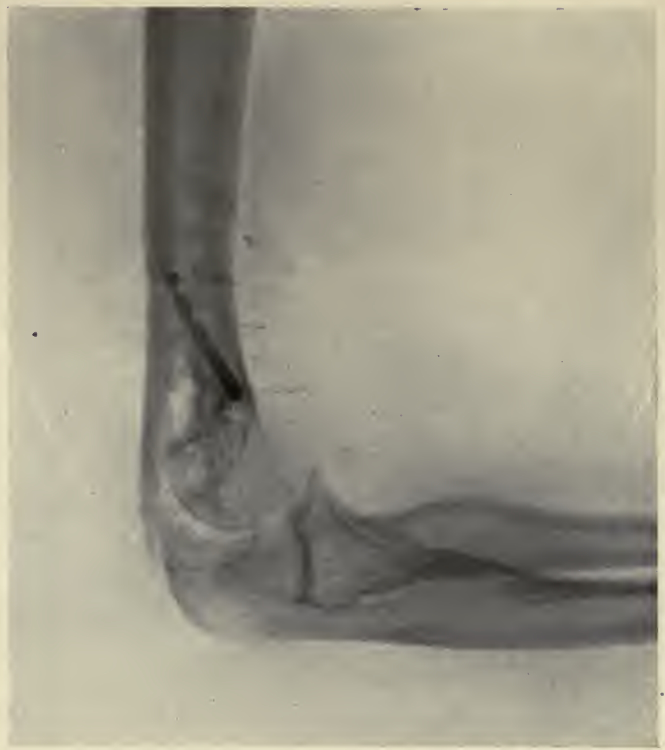


Fig. 214.—Postoperative skiagram of fracture of internal condyle of humerus. Upward displacement of condyle reduced, and reduction maintained by inserting a drill and leaving the drill *in situ* (lateral view).

union. This stage is too early for union because the neck of the internal condyle consists of compact bony tissue. It is *not* cancellated bony tissue. That misinterpretation is one of the misleading features about the duration of repair of fractures at the lower end of the humerus. Non-union hardly ever occurs in fractures through cancellated tissue nor in fractures through the extremity of the diaphysis of a bone, that is, through the metaphysis, with the exception of the location under discussion—the lower extremity of the humerus—where there is compact bony tissue. When you look at this anatomic specimen (of the lower extremity of the humerus) you see that there is no cancellated tissue in the neck of the condyle but compact tissue [indicating].

Now then, this internal condyle is not fractured only at but is also displaced upward (Fig. 215, *A*). What we want to do is expose the internal supracondylar ridge of the humerus, keeping anterior to the ulnar nerve, and endeavor, by utilizing the head of the radius as a fulcrum against the capitellum, to pull the internal condyle down into place and then insert a drill and let the drill remain *in situ* and act as a nail to retain the condyle in its lowered position. There is no occasion for removing the drill and substituting a nail. Put in the drill and leave it in and then cut the drill off to the level indicated. By leaving the drill *in situ* you obviate the danger of accidentally dislodging the fragments by pulling the drill out and substituting a nail for it. It is possible that we shall use but one drill.

The correct management of like cases when seen early is to make a nick in the skin just large enough to admit a nail and drive the nail through the nick into the extra-articular area of the bone, never compounding the fracture, and never entering the elbow-joint. (By referring again to the anatomic specimen you see how far away you may be from the joint and still have room to insert the nail (Fig. 215, *B*).) It might be necessary for you to drive the nail directly from the external to the internal condyle in order to hold the fragment in position. In this case the fracture will have to be compounded by cutting down and exposing it, because I cannot locate the condyle on account of the infiltration present and the hyperplastic soft tissue that has formed in the neighborhood of the condyle. We believe that in fractures of the condyles of the humerus the best results are obtained by nailing, using either the single or the double method of nailing.

Fracture of both condyles with backward displacement of the lower fragment is the most dangerous fracture, from a service standpoint, that occurs in the elbow-joint. This bicondylar fracture has a twofold danger associated with it. First, there is the danger of backward displacement of both fragments; and, second, the danger of the lower end of the upper fragment of the humerus impinging against the coronoid process of the

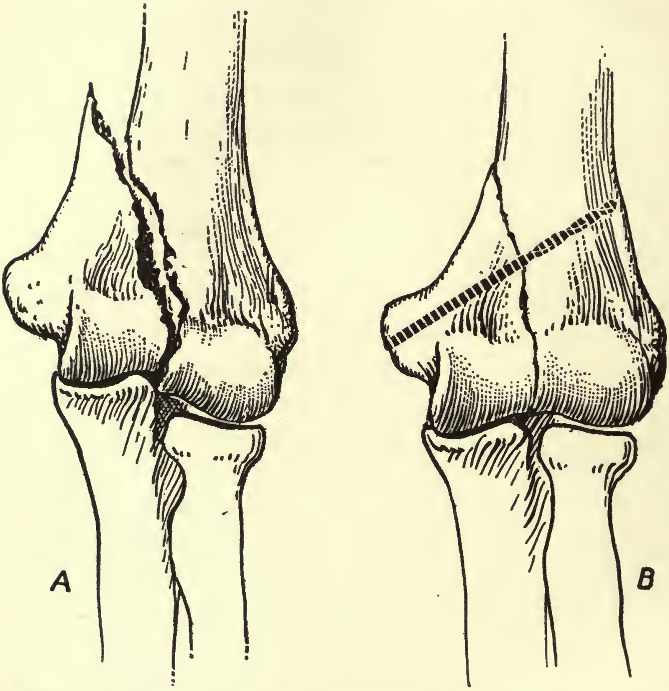


Fig. 215.—*A*, Schematic drawing of fracture of internal condyle of humerus, showing extent of upward displacement of condyle before operation. *B*, Shows displacement of internal condyle reduced and fragments secured by a drill peg. Extra-articular fixation.

ulna, so that when the patient flexes the arm the coronoid process comes up and strikes against the humerus.

Of itself, however, *backward displacement* of the fragments is not the most serious danger, but the *surgeon himself is.* Why? Because in an attempt to prevent the displacement after attempted reposition he applies a bandage so tightly that the patient suffers Volkmann's contracture. Scores—not dozens, but *scores*—of patients with elbow-fractures come here year after year, ruined by too tight bandaging. *The purpose of applying a bandage is never that of reducing the luxation of the displaced fragments of a fracture.* A bandage is of little aid in retaining the fragments in position if they have not first been placed in the proper relation by reduction. When you have a hemorrhage taking place in the antecubital fossa, invading the flexor group of muscles, if you apply an inelastic binder around the limb over the site of hemorrhage, within forty-eight hours you have ruined that forearm forever. It is shocking to think of the patients we have had who have been crippled by too tight bandaging for this type of injury. The pathologic etiology of the ischemic contracture is completely established within from twenty-four to forty-eight hours, so that even if the tight bandage were removed at the end of forty-eight hours the contracture would be just as sure to follow as though the bandage had been kept on for four weeks. The one exception to this statement which I recall occurred in the case of a patient who came from Pennsylvania, upon whose arm the bandage had been retained five weeks for a fracture of the elbow. The night following the day the injury was received the forearm became exceedingly painful and the hand puffed up. He was given morphin hypodermically three or four times a day for five weeks, while the sole cause of the pain was that these bound muscles were crying out for relief from the pressure of the too tight bandage. Instead of relieving the pain by removal of the bandage it was relieved by an anodyne, with the result that the forearm was destroyed functionally.

Nothing we can do will restore fully such a damaged forearm. In some of the cases a few of the muscle-fibers are left intact at some place, so that when we elongate the tendons after contraction has taken place we can get a fairly serviceable hand. If the destruction of the muscle is low down in its belly, close to where the tendon begins, we get a very good result and in many cases excellent results. For example, one patient was operated upon nine years after the contracture had begun, and is now an accomplished amateur pianist, a vocation the ability to resume which shows to what extent he must have recovered the use of the fingers. Another patient, a musician who was a professional harpist, suffered the same injury. Now, several months after the operation, she has been enabled to take up her concert work again.

In dressing the forearm in the early days after receipt of the injury, you dress it in a degree which is slightly less than a right angle. If that arm should become painful or the hand swollen, you must release the forearm from the position of flexion until the swelling has subsided, otherwise there might result compression and circulatory stasis and eventually traumatic myositis. Thus the same sequelæ may follow both tight circular bandaging and hyperflexion in the presence of swelling.

I remember my old associate, Dr. Lee, who had an enormous fracture service in Chicago thirty-five or forty years ago. At that time he dressed these cases of supracondylar elbow-fractures in a flexed position. Of the two directions in which the lower fragment can be displaced, a forward shifting is much less serious than a backward. Last week we had a case in which there was forward displacement. We resected the tip merely of the olecranon process, and now the patient can bring the forearm down perfectly straight. That operation was a very trifling one.

OPERATION

I should like to call your attention to a situation which you can see shown very clearly here. This lower fragment of the humerus moves in company with the olecranon process and thus with the forearm when flexed or extended. That is where non-union of the condyle occurs—at the site of compact bone.

[After bringing down the fragment and then applying a constrictor, an incision was made and the operative field developed.] After four weeks' duration of the process of hyperplastic infiltration of the tissues I am loosening this fragment and endeavoring to bring it down to its normal level by utilizing the tip of the external epicondyle as a fulcrum. Now I shall cut down at this site to see if I can insert the nail which is to hold the fragment *in situ*.

I have the line of fracture clearly in view. See the perfect alinement of the fragment! That proved to be an unusually easy exposure to make. The key to the successful completion of this bone work is the performance of the operation without hand-contacting. [Constrictor removed and wound closed.]

RECAPITULATION

Let the record show that there was less bleeding after the constrictor had been removed than there was while it was still on the arm; that the site of fracture was easily exposed; that the ununited condylar fragment was brought down into position before the incision was made; that after the incision had been made we first sought for and found the zone of the ulnar nerve, keeping close to the periosteum, and then exposed the site of fracture and held the fragments in perfect apposition, fixing them with a single drill.

Let the record show further that a single drill was passed through the neck of the internal condyle into the shaft, or metaphysis, of the humerus above the olecranon fossa (Figs. 214, *B*, and 215, *B*); that all the work was performed without hand-contacting and also without contacting with the fragments of the portion of any instrument that had contacted at any time with the hand.

A dry dressing was applied, but no collodion was used.

[*N. B.*—The patient made an uneventful recovery. When seen on February 19, 1915, one month after operation, she had good motion in the arm. There was no contraction, nor other evidence of traumatic myositis.]

[To be compared with this case is the case of "Ancient Fracture of External Condyle of Humerus; Division of Musculospiral Nerve," which was published in the CLINICS for August, 1915, pp. 679–690.]

ANCIENT FRACTURE-LUXATION OF ELBOW-JOINT—RESECTION—ARTHROPLASTY

Summary: Clinical history of patient; comments—physical examination of left arm; outline of plan of operation; chances of obtaining a movable joint in this case; importance of removing the periosteum when performing arthroplasty; bad effects of forced motion after a recent injury to a joint; importance of no hand-contacting; development of bone-ends and interposition of a flap; recapitulation.

HISTORY

THE patient, a male aged thirty-one years, was admitted to hospital on April 5, 1915. The family and past histories are negative.

The present injury was received eleven years ago, in November, 1904, when the patient slipped on an icy pavement and fell to the left side; as he fell he put out the left hand to break the fall. He succeeded in breaking the fall, but as the hand struck the ground he felt a snap at the left elbow, although the elbow itself did not strike the ground. The forearm seemed to be lifeless, and he was powerless to move it. A physician put the arm in a sling. The patient made no effort to move the forearm for a month. When at last he did try to move it, he found that he could not extend the forearm completely, nor could he flex it beyond a right angle; pronation and supination were not seriously impaired. Another physician said that the forearm was luxated. He anesthetized the patient and made an attempt to reduce the luxation, but failed. The patient than gave up hope and sought no further advice concerning the elbow-joint for nine years. Nine months ago, in July, 1914, he was operated upon in Washington, D. C., but with no improvement.

At the present time the forearm cannot be extended fully, and the range of flexion is very slight. The motion of supination is slightly impaired, and that of pronation is but little, if at all, affected. There is marked crepitation in the elbow-joint during motion. The internal and external condyles and the olecranon process deviate from their normal relations.

COMMENTS

Dr. Murphy (April 7, 1915): The range of flexion and extension of the elbow, taken at the site of the styloid processes, is only four inches, which you see is practically nothing (Fig. 216). He is a waiter by occupation, and by the accident is deprived of his means of livelihood. There are both a fracture and a luxation—there is not a luxation or a fracture alone, but a fracture and, in addition, a luxation. If you will look at that *x*-ray picture (Fig. 217), you will note that there is a fracture of the internal condyle and a backward displacement of the condyle with the olecranon process; that this process is far behind its normal relation to the lower end of the humerus; that the outer portion of the lower end of the humerus is displaced forward to an extent that a line drawn through the middle of the shaft of the humerus would fall directly upon the tip of the coronoid process of the ulna. Remember that in the normal condition a line drawn through the middle of the shaft of the humerus falls a little behind the deepest portion of the olecranon fossa, and a little nearer to the tip of the olecranon than to the tip of the coronoid process.

From the skiagram it appears to me that the patient has a fracture with a backward displacement of the internal portion of the articular surface of the humerus, so that when he attempts to flex the forearm the coronoid process strikes against the humerus and prevents his flexing the forearm (Fig. 217). It is a very unpleasant case to handle. In performing the operation I would like to divide the olecranon process and then remove the exostosis that has developed upon the displaced fragment of the humerus, but if I cannot carry out that procedure without a freshened bony surface on the olecranon fossa and on the lower end of the humerus, then I shall have to perform arthroplasty, which makes the situation still worse.

What I shall try to do is divide the tendon of the triceps muscle instead of the olecranon process, dividing the tendon

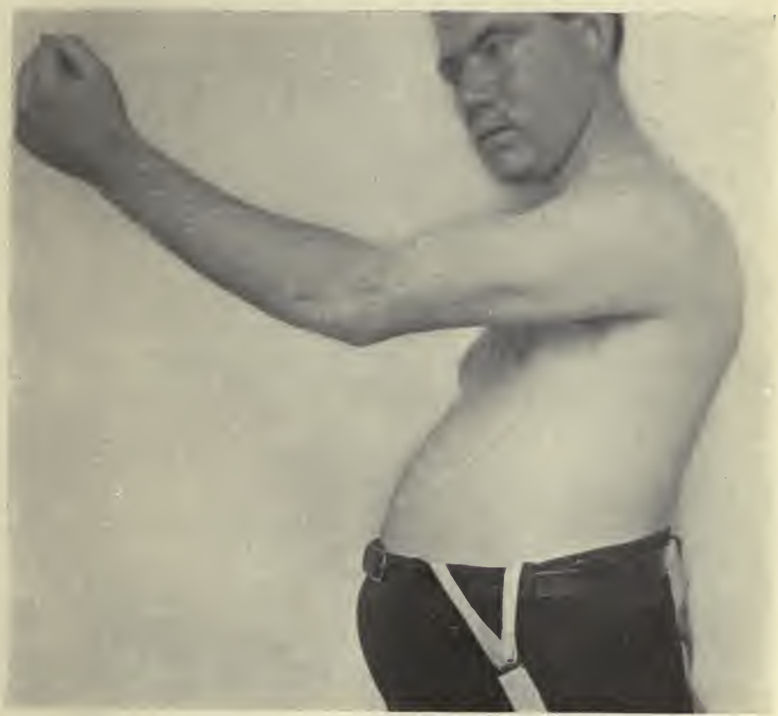


Fig. 216.—Ancient fracture-luxation of elbow-joint before operation. Range of flexion and extension of elbow taken at site of styloid process is only four inches (lateral view).

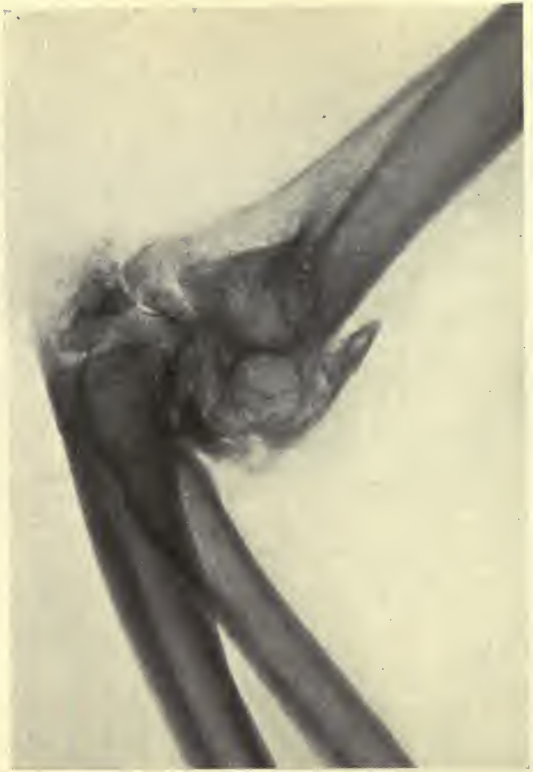


Fig. 217.—Preoperative skiagram of ancient fracture-luxation of elbow. Note backward displacement of the olecranon with internal condyle; also note external condyle resting upon neck of radius (lateral view).

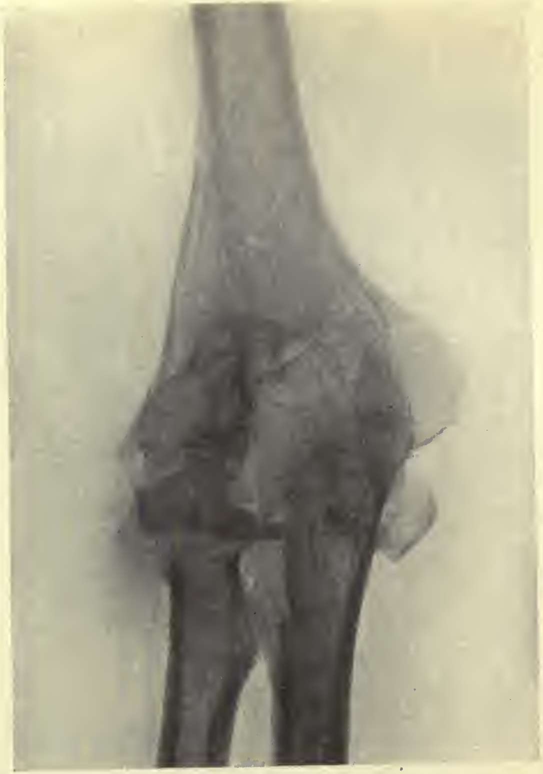


Fig. 218.—Preoperative skiagram of ancient fracture-luxation of elbow. Note displacement of radius and ulna backward and slightly upward; also note comminution of lower end of humerus (antero-posterior view).

and separating it from the bone in an L-shaped manner, so that I can unite, contact, or shorten the tendon just as I desire without exposing the joint to infection. I brought in the *x*-ray pictures made by Dr. Hochrein, which show the arterial circulation in the neighborhood of the elbow-joint, and they reveal a free collateral circulation, so that I can make the flap with the base either above or below. As I am going to divide the tendon of the triceps at this point, I shall first divide the skin below, so that the division of the tendon and of the skin will not fall in the same vertical plane. Then I shall endeavor to go in on the inner side and reach the joint posteriorly and then displace the fragments situated in front of the joint with the greatest possible precaution against injuring the structures that are situated anteriorly, working close to and pressing upon this displaced fragment and in this manner laying the bone wide open, because if I grope around anteriorly in the dark without keeping close to the bone, I might injure the brachial artery or the median nerve, either of which accidents would be serious. After making the elbow-incision the next step is to release and throw out of the field the ulnar nerve, which is in an exposed position, so that upon dividing the triceps tendon I shall not have the ulnar nerve near the path of the knife.

I have not promised this man so favorable a result as I would have promised him had he had bony ankylosis. In the latter case I could go in and perform an ordinary arthroplasty and promise him—since the chances are 90 per cent. in favor of it—that he would have a good, flexible joint. In light of the existing conditions his chances of getting a movable joint are only 80 per cent.

In removing these fragments of bone it is essential that we remove the periosteum still attached to the bone, although the freeing of the periosteum from the displaced fragments would be much easier. If I could take the periosteotome and free all these tissues still attached to the periosteum by working subperiosteally, there would not be the same degree of danger of injuring important structures as when I have to separate the periosteum from the tissues and leave it on the bone. If I were to leave the periosteum within this space, it would fill up the space with newly formed bone.

I shall show you a case in point in a patient who had an arthroplasty performed outside of Chicago. In performing the operation the surgeon elevated the periosteum with the overlying tissues while removing the bone. The result was that after the joint had healed the patient had more bone than there had been before, and consequently a greater area of ankylosis. It is only a small point in the technic, but nevertheless a very important factor in the ultimate result. Here is another case in which all the periosteum likewise was elevated from the bone: note the small range of flexion and extension. After the second arthroplasty for bony ankylosis the patient had full flexion and extension.

I have endeavored to call attention to the importance of removing the periosteum with the bone a number of times in the Clinics and in other publications, but notwithstanding I still see cases that have had the so-called "Murphy" operation for arthroplasty, but where the periosteum was not removed, and an evil result followed. It is because of the tearing of this periosteum and leaving of shreds of it behind that we have so many times large deposits of bone around a joint after operation. It is because of the trauma and the hematoma following the original injury that similar exostoses form after the accident.

In addition to leaving the periosteum there is the undesirable feature which we have emphasized for years, and are still talking about, and that is the early forced motion that is ordered for these cases of fracture in the neighborhood of a joint. When you produce forced motion in a joint where you have had a fresh wound, what do you do? If you had the joint open and could look into it and see the effects of the forced motion, what would you find? That you were tearing the tissue within the joint open. That means what? That you have hemorrhage into the joint, which will be followed by hyperplastic inflammatory tissue formation, then the formation of cicatricial tissue, and finally ankylosis. Therefore, early passive motion after fractures is always liable to impair subsequent motion. It was a terribly hard lesson for us to learn, because we had been forcing the passive motion for so many years. I remember when I was a youngster in medicine and associated with Dr. Lee that I could tell when I got within a half-block of my office that there were cases of fracture of elbow waiting, from the screaming heard when he forced the motion of the joints. It is better to wait four or five weeks until union and repair of the soft parts is complete before beginning the motion, and then begin with mild passive motion, and never with severe. Passive motion that is severe enough to cause intense pain indicates traumatic laceration of the tissues.

Let us examine the elbow again. There is the head of the radius, subluxated forward; there is the olecranon process, whose tip is broken, and here is the triceps tendon. In doing this work we endeavor to perform it throughout without touching the hands to the wound, notwithstanding that we wear gloves, and all our other precautions. In the large percentage of cases of operations for bone transplants and in arthroplasties we find that we have been getting very much better results where there has been no hand-contacting.

OPERATION

Here is the line of incision from the previous operation, and it is the line that I am going to follow. I am placing the incision here because there is present a line of cicatricial formation. Now you can see that I have not encroached upon the line of blood-supply.

The next step is the exposure of the ulnar nerve behind the internal epicondyle: there it is, you see; we displace it forward. There are the articular filaments of the nerve and we cut them off: now we are through with the ulnar nerve. We shall know where it is situated during the remainder of the operation. The nerve is the structure that is exposed to much danger when working in the region of the elbow-joint. Here is the triceps tendon. Now I am dividing the capsule of the joint at the tip of the olecranon process so as to facilitate shifting of the ulna forward. There is the external condyle of the humerus. There is the capitellum—what is left of it. Here, you see, the greater sigmoid fossa is practically filled up with newly formed connective tissue and some deposits of bone (Fig. 221, *A*). There is the internal condyle. There is the wrecked lower extremity of the humerus. Here is the fracture of the tip of the internal epicondyle united by fibrous union. Here is the fragment of the internal condyle that is displaced forward. These findings correspond to our interpretation of the skiagram, although it is not easy to obtain an idea of the real situation from the skiagram alone.

There again is the fragment that is displaced anteriorly. I must get around it and free it so as to excise it. Remember, the brachialis muscle lies here as a guide to protect you from injuring the nervous and vascular structures that lie in front of the muscle. There is another detached fragment with just a fibrous union. You see revealed here a condition that does not show in the skiagram—the seemingly close proximity of the coronoid process to the tip of the olecranon process, due to the exostosis, which is either the sequence of the fracture, or the result of some other cause lessening the long axis of the greater sigmoid fossa. There is a forward luxation of the head of the radius. I want to utilize the articular surface of the latter, so I shall take off this posterior portion of bone. There is the olecranon fossa. The humerus now looks almost normal. The next step is to scoop out the greater sigmoid fossa, and that is a situation in which you would like to use your fingers. This loose strip is a portion of the capsule, and I am whipping it over to cover the joint-surface (Fig. 221, *B* and *C*). There is a fragment of bone that must be removed.

The bone-ends now come into very good apposition, and the normal configuration of the joint has been restored (Figs. 219 and 220). Next we swing around the muscular interposing flap over this portion so there will not be left an exposed bony surface. The only exposed bony surface left now in this joint is the one on the under surface of the olecranon process: now that surface is covered. We must remove a portion of the tip of the internal epicondyle. I am leaving the ulnar nerve entirely outside of its groove, and displaced forward, for if left in the groove it

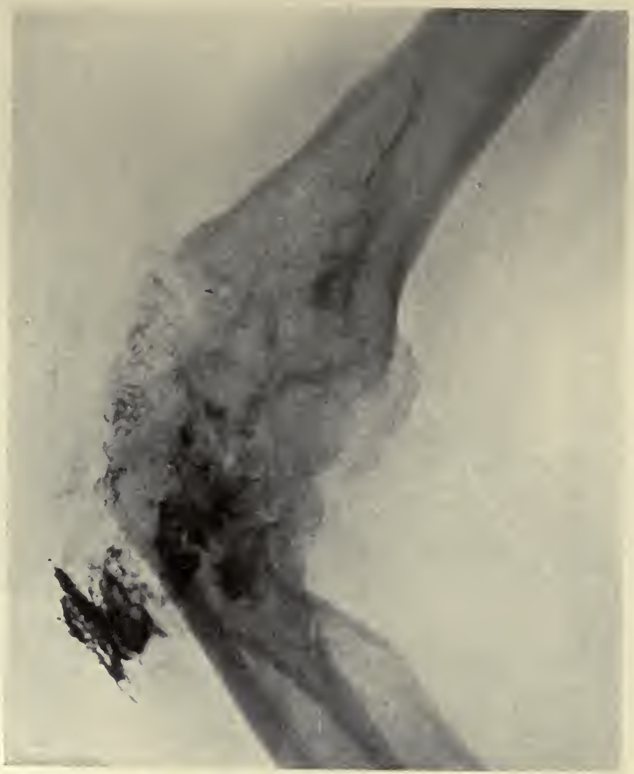


Fig. 219.—Postoperative skiagram showing relation of bones of elbow after reduction of fracture-luxation. The olecranon process now occupies its normal position (lateral view).

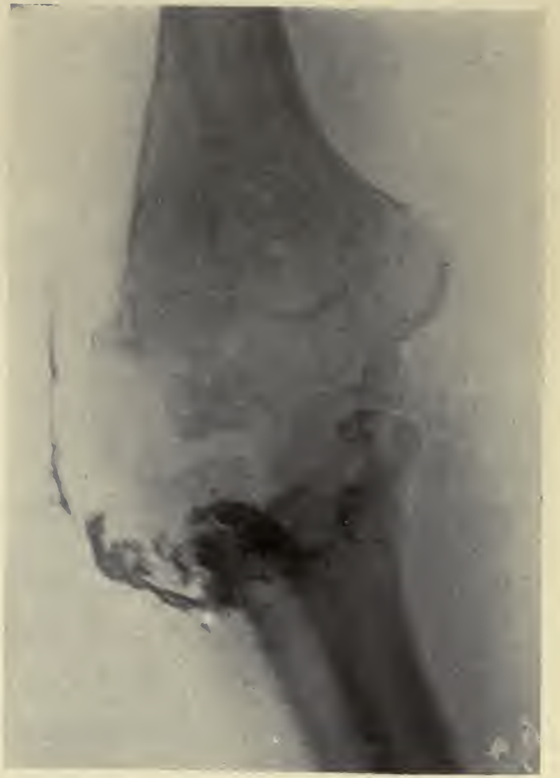


Fig. 220.—Postoperative skiagram, showing relation of bones of elbow after reduction of fracture-luxation (anteroposterior view).

will become involved in the scar. There is no raw bony surface left remaining at the site of the capitellum.

I feel very much pleased that I could perform this operation in place of the one requiring division of the olecranon process, for had I divided this process I would have made a failure of the whole procedure on account of the freshened end that would have been left. The fracture at the tip of the olecranon process tore up and elevated the cartilage lining the greater sigmoid fossa, a blood-clot formed, and the fossa filled in with bone (Fig. 221, *A*). You see how the ulnar nerve stays just where it was put—away from the humerus, between the subcutaneous tissue and the antecubital fossa.

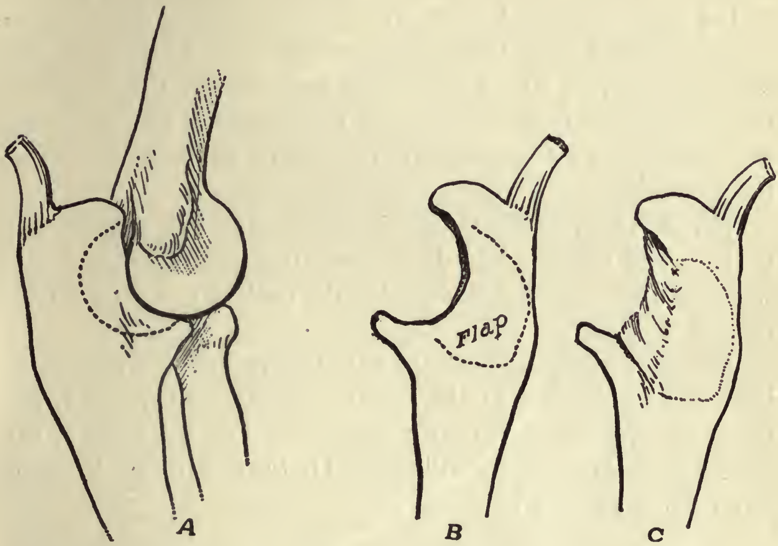


Fig. 221.—Sketches made at the operating-table of the condition of the greater sigmoid fossa of the ulna at the time of its exposure, and how the condition was dealt with. *A*, Shows greater sigmoid fossa filled with a bony deposit. The dotted line shows the extent to which the greater sigmoid fossa was restored by removal of the bony deposit from it. *B*, Shows the fossa after the bone had been removed. Dotted line indicates conformation of periarticular pedicled flap for interposition in the humero-ulnar joint to prevent ankylosis. *C*, Interposing flap reflected upon its base as a hinge across restored greater sigmoid fossa so as to form a new articular lining for humero-ulnar joint.

RECAPITULATION

Let the record show that after removing the loose fragment of bone we were able to retain the articular surface of the humerus, and after removing the mass of bone that filled in the greater sigmoid fossa (Fig. 221, *B*) we were able to get a fairly good adjustment of the bone-ends; that then the freshened bony surface of the greater sigmoid fossa was covered over with a flap of fascia obtained from the tissues on one side of the olecranon process (Fig. 221, *C*), so that there were no raw bony surfaces contacting with each other at the completion of the operation; and finally that we succeeded in performing all the work without hand-contacting.

[*N. B.* (May 28, 1915, seven weeks after operation)—The patient was examined to-day. Pronation and supination are well executed. At the elbow-joint the patient has perfect freedom of motion.]

[Cases in which arthroplasty of the elbow has been performed have been published in the CLINICS for December, 1912, pp. 833–841; December, 1913, pp. 999–1010; and the steps of the formal operation have been illustrated in the CLINICS for October, 1914, pp. 1027–1034.]

ANCIENT FRACTURE OF ELBOW-JOINT (OLECRANON PROCESS)—RESECTION OF OLECRANON PROCESS

Summary: Clinical history of patient; comments—fracture of olecranon process of ulna of lesser moment in a woman than in a man; conservative *vs.* operative treatment in this case; fractures at elbow should be dressed at first not in extreme flexion; danger of too tight bandaging; operation—removal of fragment of olecranon process restores power of full extension; no hand-contacting technic in joint; importance of removing periosteum with bone to avoid callus-formation; no passive motion of joint before fourth or fifth week; embryology of joints.

HISTORY

The patient, a female aged twenty-three years, was admitted to hospital December 30, 1914. The family and past histories are negative.

The present injury was received over seven months ago, on May 9, 1914, when the patient was in a runaway accident. She was thrown out of the buggy, striking upon the posterior portion of the left elbow. A physician immobilized the left wrist—but not the elbow-joint. The forearm was put in a splint that extended from the finger-tips to the elbow. This splint was retained three weeks. On the night of the accident the pain was very severe. At the time of the injury all sensation in the hand and forearm was suspended, but returned an hour later. Since the accident she has had more or less pain and is occasionally awakened nights by it. The pain is more severe when an attempt is made to move the forearm and also during cold weather. Since the splint was removed she has had no trouble with the wrist.

The patient now has a good range of motion in the wrist-joint. The elbow is still painful, and she is still unable to extend or fully flex the elbow-joint. The movements of supination and pronation cause severe pain, as well as a catching of the elbow. Over three months ago, on September 12, 1914, a skiagram was taken, and showed that the wrist was normal, but that there was a fracture of the olecranon process of the ulna.

COMMENTS

Dr. Murphy (January 2, 1915): When you look at that *x*-ray picture what do you notice? You notice that the tip of the olecranon process does not pass behind the posterior plane of the humerus; normally it is situated about one-third of an inch back of that plane. You see that the tip of the olecranon process strikes against the lower articular end of the humerus, and thus blocks extension of the forearm (Fig. 222). This is a beautiful illustration of that anterior relation of the olecranon tip. Here is the tendon of the triceps attached to the olecranon process as far down as its lower margin. There is the subcutaneous bursa over the tip of the olecranon process, and all the fibers of the triceps muscle that afford leverage are pulled downward. You may, therefore, take off all the so-called anterior tip of the olecranon process and still not impair the power of extension of the forearm.

You know that in a woman the power of extension of the elbow has very little value. Woman is not built for pushing people away from her, but for "pushing" people toward her. If we can get this elbow-joint relatively straight, that much extension will suffice, for she does not need complete power of extension. We talked this matter over very frankly with the patient and with her physician. The physician got a good result from a very bad condition—in fact, the best result that could have been obtained by conservative measures. The reasons why this was a difficult fracture to treat without nailing are, in the first place, because it was such an extraordinary level at which to have a fracture; and, secondly, because there is no control over the lower fragment at all. Now let me show you the range of motion in this elbow-joint. That is a mighty good arm because she can put her hand up to her head to feed herself and to arrange her hair, but the elbow is not so straight as she would like to have it because the tip of the olecranon process strikes against the lower end of the humerus: that is what we are going to try to correct.

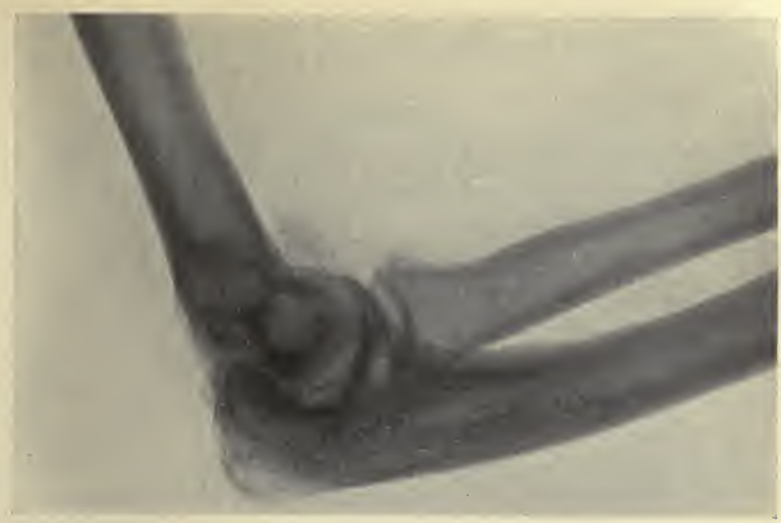


Fig. 222.—Preoperative skiagram of ancient fracture of tip of olecranon process of ulna. Note how tip of olecranon process strikes against lower end of humerus, thus blocking extension (lateral view).

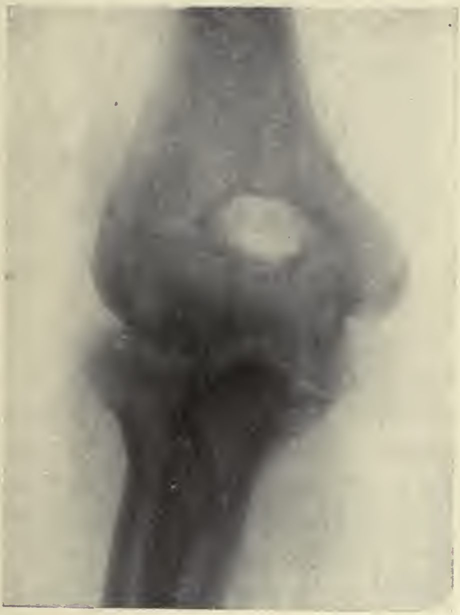


Fig. 223.—Preoperative skiagram of ancient fracture of tip of olecranon process of ulna (anteroposterior view).

OPERATION

[Incision.] We are going in from the radial side because by that avenue of approach we do not have to bother about the ulnar nerve, which is behind the internal epicondyle. There is also a fragment detached from the head of the radius.

Now, you see, there is the margin of the olecranon process, to which the tendon of the triceps muscle is attached. There is the site of the subcutaneous bursa: the fracture is just below the tip of the olecranon process. There is the opening into the elbow-joint. There is the fragment of this olecranon process, and it must be removed. We must remove the periosteum with the fragment; if we do not, by periosteal proliferation the limitation of motion will become worse than it was before the operation. Now the forearm comes straight. The removal of that fragment of the olecranon process is just what was needed: you see the spot where the fragment struck against the humerus and interfered with extension. There has been practically no trauma to the elbow-joint. We did not touch the joint with anything—not even with a sponge. You remember how many years it took us to learn to keep our hand, gloves, and instruments from traumatizing the peritoneum; as soon as we learned that important lesson we had sepsis no longer, nor did the patients leave the table comatose.

RECAPITULATION

Let the record show that it was clearly discernible in the open field where the fragment of the olecranon process struck against the posterior surface of the humerus, and after removal of the offending fragment we were able to bring the forearm down to a straight position, and that one-half inch of the tip of the olecranon process was taken off without traumatizing and without injuring the joint, or even touching the joint with the portion of any instrument that had previously touched the hand.

POSTOPERATIVE COMMENTS

The patient will not develop callus because we took the periosteum off with the fragment. If you peel off the periosteum and leave it behind after removing the fragment of bone, then you are liable to get callus-formation. That is the source of mischief in fractures of the elbow. If you start passive motion early, a hematoma forms because of the manipulations and later you get a soft callus which finally becomes ossified (see previous case). Every time you begin passive motion before the sixth week you increase the amount of callus. We have just learned that in a fracture near a joint passive motion must never be begun before the end of the fourth or the fifth week. If you had a cut on the forearm and kept pulling the edges of the wound apart every other day, what would you have in the end? The wound would finally heal of itself, but you would have left a mass of connective tissue. If you get connective tissue near a joint much of it ossifies. That is what produces ankylosis. Remember that in the early stages of the embryo there are no joints. The forerunner of a joint is just a mass of cartilaginous tissue. The next step in the evolution is the cleavage of the cartilage into a joint-space, and that tissue which was originally the perichondrium eventually forms the fibrous capsule of the joint.

[*Note.*—Complete primary union and a good functional result ensued.]

FRACTURE OF RADIUS AND ULNA—NON-UNION OF RADIUS—INTRAMEDULLARY AND INLAY TRANSPLANTS

Summary: Clinical history of patient; comments—further elucidation of history; Lane plate not intended for the treatment of non-union; deformity of ulna may be ignored and attention focused upon radius; operation—incision between and *not into* muscle-groups; skin excluded from operative field; disposition of periosteum of a transplant; intramedullary implantation with inlay bone-grafts outside; recapitulation; postoperative comments—importance of immobilization after insertion of transplant; non-union after fractures of radius becoming very common; hematoma stimulates osteogenesis if in a sterile field; hemorrhagic cyst as a cause of non-union; non-union liable in fracture through neck of a humeral condyle owing to presence of compact tissue; postoperative treatment; prognosis.

HISTORY

THE patient, a female aged twenty-eight years, was admitted to hospital December 15, 1914. The family and past histories are negative.

The present injury was received over eight months ago, on April 6, 1914, when, in stepping from a motor, the patient caught her skirt and fell, striking her forearm upon the curb and sustaining a compound fracture of the radius and ulna. A physician set the limb and dressed it upon a splint. At the end of three weeks the splint was removed and a gypsum case was applied and retained five weeks. On removing the gypsum case it was found that the patient was able to pronate and supinate the hand but that the forearm was painful. Several days later she consulted another physician who said that the bones were not yet in good apposition. A skiagram taken at this time, at the end of six weeks, showed soft callus-formation around the bone.

Nine weeks after the occurrence of the accident, on June 9, 1914, the patient was operated upon and a Lane plate applied to the radius. Since July, 1914, the sinuses in the forearm have been healed. At no time was there any discharge.

COMMENTS

Dr. Murphy (December 16, 1914): What is lacking in that history? What happened to the compound portion of the fracture? There are no data as to when it healed, or as to whether it healed primarily or secondarily. Did she have suppuration complicating the fracture? Were both bones compounded or only one? Was there an open wound when the physician inserted the Lane plate? That history has no value because practically everything else of importance, except the fact that a plate was put in, is left out. Did the wound suppurate? That is the first thing to ask about a compound fracture. Was there good primary union? I am sorry to have to ask these questions in such a way, but they should have been answered in the history. The patient's statement is that union of the ulna *had* occurred, and that for non-union of the radius a Lane plate had been inserted.

You will recall that one of the things about non-union Mr. Lane accentuated is that the plate is *not* intended for the treatment of non-union of a fracture: he made that statement in his address on this subject right here in this arena. Whether the fracture was compounded at the time the plate was inserted or whether it was compounded primarily we do not know, but if there was an open wound at the outset, it remained healed after the plate was inserted, and has not discharged since the operation. For that reason the plate is fully covered, as you see. The physician who inserted the plate did his work well, because the plate remained in, and no suppuration followed. There is some deformity (Figs. 224 and 225). There is union of the ulna, but you see that the deformity of the ulna is not great in extent. We have trouble enough at this time in trying to secure union of the radius, without refracturing the ulna.

OPERATION

[Incision.] I shall follow as nearly as possible the line of division between the flexor and extensor groups of the muscles

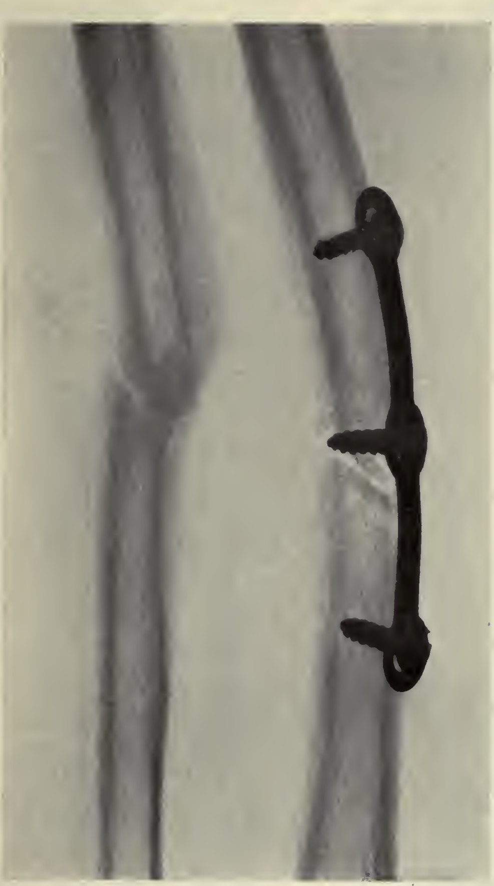


Fig. 224.—Skiagram showing fracture of radius and ulna. A Lane plate had been inserted into the radius elsewhere. Note how central screw passes through line of fracture (anteroposterior view).

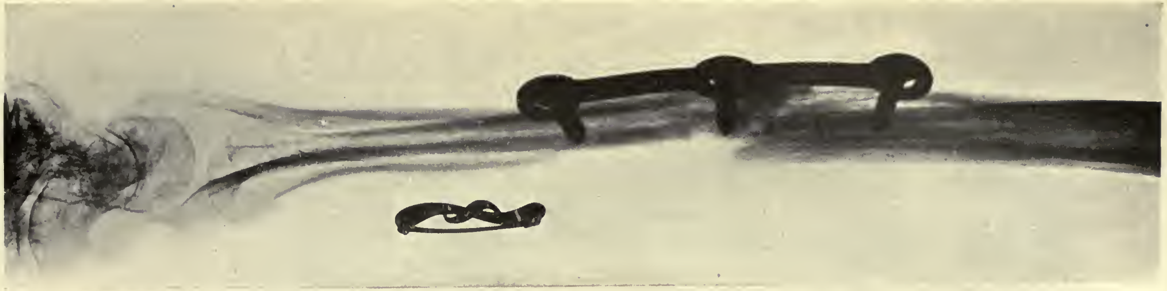


Fig. 225.—Same as Fig. 224 (lateral view).

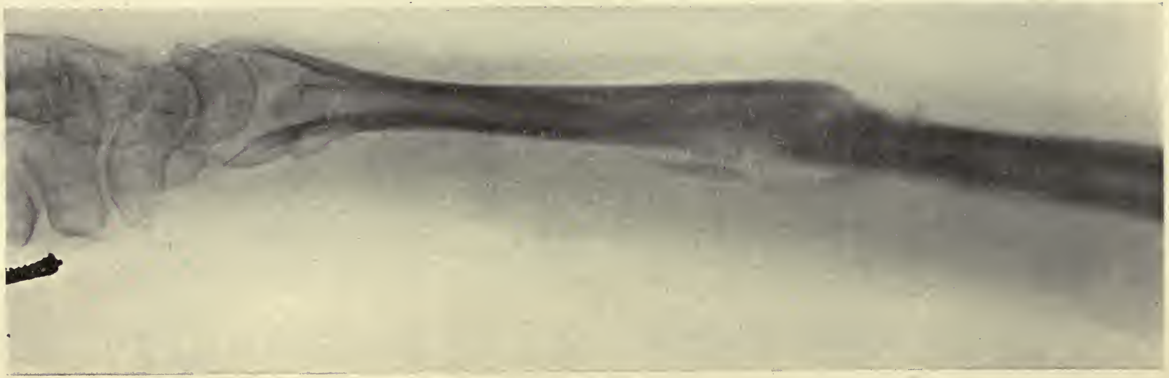


Fig. 226.—Postoperative skiagram of fracture of radius and ulna after removal of plate from radius and insertion of intramedullary bone-splint and bone-graft inlay (lateral view).

of the forearm. We never cut directly into either of these groups of muscles if it is possible to avoid it, as the ensuing fixation of the muscles to the bone is very annoying to the patient. We endeavor to cover the edge of the skin with towels, so as not to have the skin exposed in the field at all.

Let the record show that there is no hematoma and no exudate about the plate.

This lowest screw was apparently put into the line of fracture, and it has remained firm. The central and the highest screws are loose and do not have to be unscrewed to be removed, since the bone around them is soft. There is the line of separation of the bones. Peeling off of the periosteum for a considerable distance above and below the line of fracture we believe to be an important factor in stimulating osteogenesis—not peeling it far enough to cause necrosis of the bared bone, but far enough to cause the formation of a hematoma beneath the periosteum. I want to call your attention to the fact that there is no effort at osteogenesis here, as there is no callus. This patient is very light in weight, and you see how small the radius is.

I think we can employ a better plan in this case by using a transplant. I think I can invaginate this distal fragment into the medulla of the proximal. Let all the bone-chips that you take off remain, because they are of value as separate foci of osteogenesis. You can see that the fragment of bone that is available to put into the medulla is so small that of itself it would have but little strength, and but little supporting power. I think I shall have to use a transplant in addition to the intramedullary splint. I shall first freshen this medulla.

Now we shall take a piece from the tibia—a piece which necessarily will have to be very small. In removing the bone-graft from the tibia, always place your incision over the tibialis anticus muscle instead of over the shin. You see what a very small piece of bone I need insert here. We shall leave these separated fragments in because we need them all as foci of osteogenesis. That was delicate work, but it has come out very well. It was the most delicate bone-transplantation we have had to perform up to the present time.

RECAPITULATION

Let the record show that we first exposed the plate (Figs. 224, 225) and then removed it; that the central screw and the highest screw were loose; that the lowest screw was firm in the bone, and absorption had not taken place around it; that the loose fragment of bone was extremely small; that we had difficulty in making the medullary cavity large enough to accommodate the bone-graft; that we made an implantation into the medullary cavity; that in addition to that we put two transplants outside so that we have both a medullary splint and cortical splints (Fig. 226); and finally that the skin was closed with catgut.

Let the record show further that all this work was accomplished without hand-contacting of the wound or of anything that had touched the wound.

POSTOPERATIVE COMMENTS

With a bone-graft you need absolute immobilization if you are going to get a good result. In fractures of the shaft that are treated without a transplant it is essential to have some slight motion. In fractures near a joint it is unnecessary to have motion because near a joint you have osteogenesis taking place without any motion being provided for, and you rarely ever have a failure of union except at the neck of the internal and of the external condyle of the humerus. In fractures involving the shaft you can have immobilization so complete that no osteogenesis will take place, since with no motion there is not enough stimulation. This matter of mobilization is important when you have both ends of the bone living and actively osteogenetic. It is an entirely different matter in the case of a transplant. With a transplant you want the blood-vessels of the living receptive bone to pass into the Haversian canals of the transplant without the formation of connective tissue between the graft and the bone. What happens if you keep moving the fragments after insertion of a transplant? You finally have a fibrous capsule develop over the transplant with resulting failure of union. The forearm is the most difficult portion of the body to keep absolutely immobilized. In order to immobilize the radius the fixation must extend from the forearm clear up to the shoulder, otherwise there will be failure of union.

Of all late fractures in the upper extremity we receive, the most common failures of union occur in the radius and ulna. Formerly we had most commonly failures of union in the middle third of the shaft of the humerus, and in the lower third less frequently than in the upper third.

You see what will happen unless this hand and forearm be perfectly immobilized. The bone-graft will be elevated and that slight degree of elevation will prevent union of the fragments. We have, however, inserted both an internal medullary and an external bone-graft splint, and have elevated the periosteum, so that we have the best possible conditions for obtaining union. When you elevate the periosteum, a hematoma forms beneath it, and that hematoma stimulates osteogenesis, except in certain conditions in which infection has occurred. In the presence of mild or attenuated infection the hematoma breaks down as a clot and is detrimental to union.

That was one of the common causes of non-union which we found as long ago as in the eighties, and I described it in an article which appeared at that time. It was a hemorrhagic cyst that formed between the ends of the bone and became encapsulated, preventing union. It had an endothelial lining which did not admit of absorption of the bloody fluid. The fluid assumed the character of the bloody synovial fluid which is occasionally seen in the pseudo-arthroses. What the factor is in the presence of a hemorrhagic cyst that prevents the granulation tissue from penetrating and absorbing the blood-clot, nobody has yet found out. We have a case of fracture of the femur with similar hemorrhagic cystic formation and non-union in this hospital from Des Moines, Iowa, in which the physician is being sued for malpractice on the ground that the fracture had not been properly treated. The doctor was not in the least responsible for this non-union. We have an identically similar case in a physician's son from the North Side of Chicago with the same type of hematoma: his fracture had never been compounded. This boy had a hemorrhagic cyst develop in between the ends of the femur, and would never have had union without removal of the cyst. [This case was published in the CLINICS for February, 1915, p. 177, together with an illustration of the cyst *in situ*.]

The reason you have failure of union after fracture of the neck of the external or of the internal condyle of the humerus is that there is no cancellated tissue: it is all compact tissue at those sites. The lower end of the humerus thus differs entirely from the ends of bones in other positions in the body. When either condyle of the humerus is broken off you occasionally have non-union resulting. We had one case in which there was non-union after fracture of both condyles.

This dressing will not be disturbed for five or six weeks unless the patient should develop fever, indicating the presence of infection. If the operative field remains sterile, she should obtain union without difficulty.

ANCIENT FRACTURE OF RADIUS AND ULNA —DIVISION OF ULNAR NERVE—NEURORRHAPHY

Summary: Clinical history of patient; comments and operation; ulnar nerve and artery found divided; ulnar nerve involved in mass of connective tissue and shows a neuroma; contrast between appearance of tendon and of nerve; distal end of ulnar nerve found degenerated and displaced backward by styloid process of ulna; nerve-ends prepared for contacting and then approximated; nerve-ends buried in the belly of the flexor carpi ulnaris muscle as a shelter; recapitulation.

HISTORY

The patient, a male aged thirty-nine years, was admitted to hospital December 13, 1914. The family history reveals that two uncles died of carcinoma—one of cancer of the liver, and the other of cancer of the stomach. The past history is negative.

The present injury was received three years ago, in 1911. While the patient was cranking his motor the engine backfired, with the result that the right radius and ulna were broken (Figs. 227, 228). The fractures were reduced and the forearm was dressed upon a splint for six weeks. When the splint was removed he noticed that he did not have proper control of the hand. There was loss of sensation, and especially of the muscle-sense. Since that time there has been atrophy of those muscles of the hand that are supplied by the ulnar nerve.

COMMENTS AND OPERATION

Dr. Murphy (December 14, 1914): There is a mass of cicatricial tissue here which is the sequence of the injury. I feel a large neuroma on the distal end of the ulnar nerve. [Incision.] There is the neuroma coming into the field; you see that mass of connective tissue fills all this space at the site of the fracture. There is the ulnar nerve; here the ulnar artery. We are separating the vessel from the neuroma in an endeavor to follow the tract of the ulnar nerve. You see that the ulnar artery has been divided as well as the ulnar nerve. See the vessel standing out free from the neuroma! I do not wish to soil the operative field with blood, as it might increase the difficulty in finding the distal portion of the ulnar nerve. There is another mass of connective tissue which has extended into this neurovascular field, and which appears to entangle the neuroma and the distal portion of the ulnar nerve. In dealing with nerves we observe the same precautions about hand-contacting that we observe in bone and joint work. Now I have the ulnar artery on the radial side of the ulnar nerve.

Let the record show that there is a great mass of firm fibrous tissue insinuating itself between the ends of the ulnar nerve.

Now you can see the contrast between the tendon of the flexor carpi ulnaris muscle and the ulnar nerve. I want to call your attention to this contrast. You notice the glistening color of the tendon contrasted with the dull, pinkish color of the degenerated nerve. You can always tell the difference between a tendon and a nerve by the difference in their color. There is the glistening tendon, here the dull, pinkish nerve. Here is the proximal portion of the ulnar nerve and there the distal portion. Let us see if any of the axons of the distal end of the nerve are living. No; the axons do not respond to the Faradic current. That contraction which you do see is the effect of the electric current upon a neighboring muscle.

Let the record show that there is no evidence of axonal activity on the part of the distal end of the ulnar nerve.

I want you to note the direction which the distal end of this nerve takes (Fig. 227). See where it dips down here! Now the severance of the ulnar nerve, and the burial of its cut ends in a mass of fibrous connective tissue, explain why this nerve has had its transmission of impulses suspended.

Let the record show that the distal end of the ulnar nerve was shifted deep down through the effort at reduction of the bone displacement, and extended down to and beneath the styloid process of the ulna (Fig. 227).

This is a beautiful illustration of what has happened. Let me

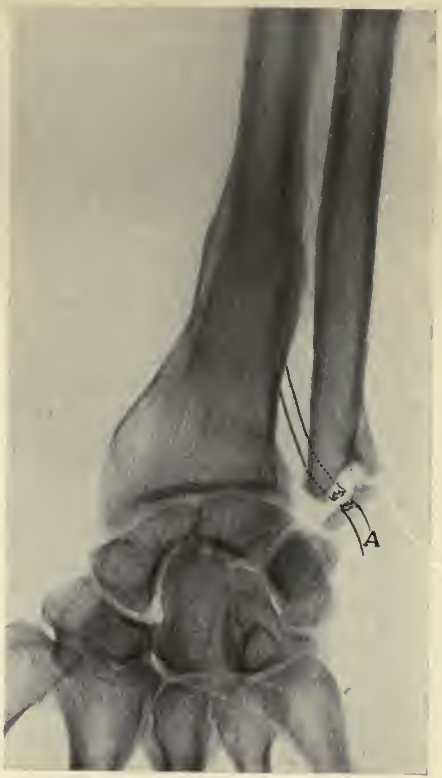


Fig. 227.—Case 9.—Skiagram showing ancient fracture of radius and ulna. The relation of the ends of the divided ulnar nerve to the fragments of the ulna is shown by an outline insert (antero-posterior view).

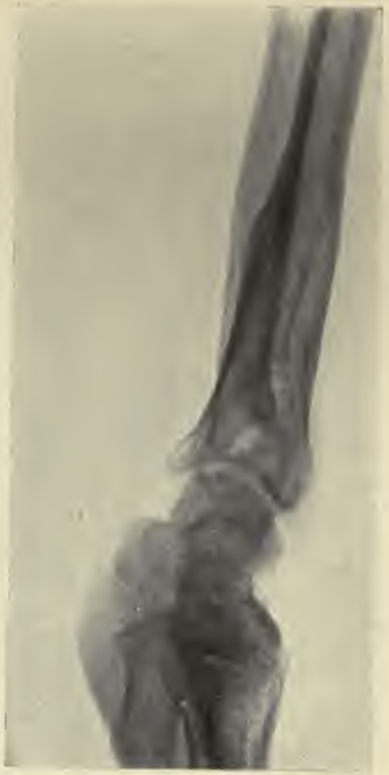


Fig. 228.—Case 9.—Ancient fracture of radius and ulna (lateral view).

show you. See that nerve! I am down upon the periosteum of the ulna. Let us come back to the upper side of the point of division and see where the proximal end goes to. You will note in the skiagram that the lower fragment of the ulna has been splintered. I take it that this nerve was not cut by a sharp fragment at the time the fracture was received, but that it was displaced down with the ulna at the time the fracture was reduced. This displacement of the distal fragment of the ulna was probably the etiologic factor in the production of the non-union which you see in the skiagram. There is where the sharp corner of the ulna caught the nerve and carried it backward. It shows here beautifully. There is the capsule of the inferior ulnar articulation. See! There is where a comminuted chip of the distal fragment of the ulna was thrown down at right angles to the long axis of the forearm. We can remove the comminuted fragment and throw it out of the field, now that we have freed the ulnar nerve from the connective tissue that bound it down.

We have seen where the ulnar nerve is divided. We shall next see if the separated ends will stretch until they come into contact. I must resect each end of the divided nerve until I can see the axons plainly bristling. Now you begin to see the fasciculi of the distal end standing out like brush-ends. We are not quite far enough back from the injured portion of the nerve yet. Now you see the fasciculi standing right out in the open. Look at them! The distal end is ready for contacting. I shall commence to work in this cicatricial mass of connective tissue and keep dividing it until we see the proximal end. There is the other end! Now the proximal end of the nerve is being developed and the axons commence to appear. There are the freed axons: see them sticking up and bristling! See how beautifully they show!

We shall shorten the distance between these two prepared nerve-ends so as to get axonal contacting. The ends will approximate with ease. My first step in effecting the approximation of the ends of the nerve is to free the proximal portion of the nerve. The traction upon the nerve is going to be chiefly upon the proximal portion and not upon the distal portion. In order to relieve all the traction on the nerve and prevent its getting back again, I take in the bite of the needle the cicatricial tissue resting upon the capsule of the neuroma on the proximal end of the nerve and bring it down and anchor it. Now I am coming into the territory upon the side of the nerve, and fix the periphery of the nerve to the nearby tissues, so that there will be no tension whatever upon the nerve.

What is the next step? The next step is to bury the nerve so that it will be out of that field of cicatricial tissue and no longer exposed to future damage by the scar-tissue. Here is the flexor carpi ulnaris muscle tendon and its fascia on the inner side. I shall just split that mass of connective tissue resting here upon the flexor muscle. Now I shall loosen the outer stitch, as this muscle affords a better shelter for the nerve than the tissues in which we had previously buried it. I shall reinsert the stitch into the fascia for support of the nerve. We shall also take off that stitch that was inserted into the connective tissue on the inner side of the nerve, because I feel that the muscle is the best place in which to bury the ulnar nerve. I shall relieve the tension upon the nerve by inserting this second stitch in this special manner. [Indicates.]

RECAPITULATION

Let the record show that with care we were able to expose the involved portion of the ulnar nerve; that the distal end of the nerve had been carried down during the attempts at reduction of the fracture to behind the tip of the styloid process of the ulna, this bony process holding the divided distal end in a position in which it could not contact with the proximal end (Fig. 227); that both ends were freed from the constricting connective tissue and resected until the fasciculi bristled clearly and distinctly; then the resected nerve-ends were approximated by both perineural sutures and direct neural sutures; that the approximated ends of the nerve were next buried in a neighboring muscle-belly so as to prevent their exposure to the open field of contracting connective tissue; and that finally the skin was approximated with catgut sutures.

The most difficult part of the operative procedure was the freeing of the nerve from beneath the tip of the styloid process of the ulna. The hand will be immobilized by a wire forearm-splint for three weeks, at the end of which time passive motion will be begun.

[*Note.*—There was primary union.]

[Of interest in connection with the above is the case of "Neuroma of the Ulnar Nerve, Result of Cicatricial Compression Following Unrecognized Fracture of the Internal Epicondyle of the Humerus," which was published in the CLINICS for April, 1914, pp. 369–374, and a second case occupies pp. 375–380.]

EMPYEMA OF PLEURAL CAVITY—RESECTION OF RIBS (ESTLANDER)

Summary: Clinical history of patient; comments—fixed sequence of symptoms in development of tuberculous pleurisy; importance of diagnosing tuberculosis before mixed infection occurs; importance of early operation in tuberculous pleurisy; a chest-sinus is easy to establish but hard to close; *modus operandi* of formalin in glycerin; beneficence of serosanguineous pus that is sterile and rich in trypsin; duration of chest-sinus after operation varies directly with duration of empyema before operation; "leather-bottle" abscess-wall in late cases of empyema; three types of thoracoplasty; operation—resection of ribs; recapitulation; postoperative comments—advantages of Delorme operation; review of subject of empyema; value of pleural effusion as nature's mode of compression of lung, and inadvisability of aspirating it; value of exploratory paracentesis thoracis; postoperative note.

HISTORY

The patient, a male aged thirty-eight years, was admitted to hospital June 25, 1914. The family history reveals that one brother died of tuberculosis of the lung two years ago. The past history discloses an attack of what was probably pneumonia, two years ago.

About eight years ago, in 1906, the patient contracted a cold and later a bad cough with malaise. Then there began sharp, piercing pains in the right side of the chest. He does not know whether or not he had fever at this time. Each attack of pain lasted from two or three days to a week and then gradually subsided until the pain finally disappeared. The patient says that he catches cold upon the least provocation, and that on catching cold he suffers from severe pain. He had three or four similar attacks in the course of a year. During the entire time the patient had a cough. Eight years ago, when the pains began, he had night-sweats for about six months and again three or four months previous to an operation in October, 1911. He says that he also had fever, but does not know how much, except that the temperature was higher in the afternoon. During the first attack of night-sweats he lost about 15 pounds in weight, and tubercle bacilli were found in the sputum. The patient continued to work throughout the course of the illness.

Almost three years ago, in October, 1911, he felt weak and had a racking cough. He consulted a physician who suggested the probability of the presence of empyema. Paracentesis thoracis revealed pus. He was advised to undergo an operation for drainage of the pleural cavity. At the operation a drainage-tube was inserted into the pleural cavity at the posterior axillary line on the right side, and one and one-half inches below the angle of the scapula. Much pus was discharged during the first two weeks, and the abscess-cavity has continued to discharge, in lessened quantity, up to the present time, the quantity now being about a tablespoonful a day.

Two months after the drainage-operation bismuth paste was injected through the sinus, but interfered with the drainage, causing elevation of temperature, and was of no benefit. Within two weeks the bismuth paste had all been expelled from the wound. Two months previous to admission, basing his opinion upon a skiagram, the physician told the patient that there was a large cavity in the right side of the chest for which an operation was indicated, and also that there were evidences of tuberculosis in the left lung.

COMMENTS

Dr. Murphy (July 3, 1914): I want to emphasize the fact that the patient did not have the pain before the cough: he had the cough first and then developed the bad pain. The cough *always* precedes the pain and the physical signs in a pleurisy of this type. The family history is one of tuberculosis, a brother having died of that disease only two years previously. Patients in whom tuberculous pleurisy develops have first the symptoms of involvement of the lung, as lassitude and fatigue, then post-meridian rise of temperature to 99° F. or 99.5° F., then the cough and finally the physical signs of lung involvement and then of pleurisy. That is the order in which the symptoms of tuberculous pleurisy appear, and their order is just as regular as that of the symptoms of an attack of appendicitis. In this case the pleurisy is the sequence of infection of the tuberculous type, which infection began in the lung.

In the very early stage of many pulmonary tuberculoses the patients have first a dry cough, which is present often for months, with practically no expectoration. When to the tuberculous infection a pyogenic is added, as an acute bronchial infection, etc., the expectoration, the night-sweats, and other symptoms and signs arising from the mixed infection appear. The sequence of symptoms is just as regular as the sequence of the malaise and the backache preceding the manifestations of typhoid fever, and as that of the symptoms of measles preceding the eruption. Tuberculosis is usually a summer disease with winter manifestations. What do I mean by that? The patients contract tuberculosis in summer, and when the fall season comes, with the pyogenic, influenzal, and other air-passage infections becoming prevalent, to their tuberculous infection is added pyogenic infection, and this mixed infection heralds the cough and the expectoration. The time to detect the presence of pulmonary tuberculosis is before the mixed infection appears, when the patients come into the office with the tired feeling, the lassitude, the languid fatigue (the "dumb ague," "malaria," "hookworm"), and with the temperature rising regularly every afternoon to 99.6° F. or 99.8° F. and rarely to 100° F.

This patient has had the cough eight years. The question arises, How long did he have the pus in the chest previous to October, 1911? It must have been a long time, for had he had pus in the chest a short time only, the cavity would have closed shortly after the drainage operation. The pus had evidently been present months or years; an encapsulated cold abscess formed in the pleura, and, when it was opened and drained, it pursued the following course. The abscess-wall, being a thickened, fibrous membrane that had inclosed the pus for years, could not collapse. The physician opened the abscess in October, 1911, five years after the onset of the trouble. The abscess cavity is still open and will remain open as long as the patient lives, unless we take steps to close it.

Any surgeon can open a chest with pus in it just as easily as he can *open* a boil, but we are looking for the surgeon who can *close* the resulting sinus. The persistence of these sinuses led us many years ago to practise injections of the pleural cavity with a 2 per cent. solution of formalin in glycerin, repeating the injections until the pus in the abscess-cavity became sterile and converted into a serosanguineous fluid, which is allowed to stay until it is absorbed. What does the solution of formalin in glycerin accomplish? First it kills the microörganisms in the pus; secondly, it keeps up a polymorphleukocytosis. What does polymorphleukocytosis indicate? It indicates an increase in the number of phagocytes and the formation of serum that contains trypsin, which is similar to the pancreatic trypsin. When the polymorphleukocyte dies it yields up trypsin. Trypsin converts albuminoids into peptones which are absorbable, and thus the process of absorption of the exudate is begun. When the pus in the pleura has been converted into a serosanguineous fluid, let it alone! It might be three months, six months, or twenty months absorbing, but the slowness of absorption does not harm the patient any because the serosanguineous nature of the fluid indicates that the microörganisms have been killed and that it is, therefore, not toxic to the patient. If the fluid can be kept in the pleural cavity without being toxic to the patient, as a sterile serosanguineous fluid, it will finally become absorbed, and the walls of the abscess-cavity will slowly contract and coalesce, and the cavity is obliterated; with this contraction the chest-wall sinks in, the lung expands, and the diaphragm on that side resumes its bellows-like activity. That plan is the only rational one of treating empyema. If you receive the case early and open and drain the abscess-cavity within the first two or three weeks of its appearance, the lung is still capable of expansion. On the other hand, if you receive the case late and then open the abscess, you get as a sequel just what was present in this patient—a chronic sinus with a permanent cavity.

The operation we intend performing this morning is of colossal extent. The very late drainage-operation should not have been performed upon this patient. Once you have opened the abscess-cavity in these late cases it will never close spontaneously. You have a "leather-bottle" to deal with, and you must treat the contents of the bottle until absorption has occurred. To this end, inject half an ounce of a 2 per cent. solution of formalin in glycerin which has been mixed at least twenty-four hours previously. That is about as high a percentage as you ever need inject, because formalin is such a powerful antiseptic that it kills the microörganisms in very weak dilutions, while if used in greater strengths it is poisonous to the patient. Venice turpentine is a more desirable drug for injection into pus-cavities where an increase in the polymorphleukocytosis is indicated, but we do not yet know how to control the reaction.

OPERATION

[Explores with probe.] This cavity is of very great extent. In removing the ribs we shall begin above at the costochondral junction and then descend until the chest-wall collapses.

In this case two ribs had been removed previously when drainage was inserted. In defending itself against contraction one rib has developed such a thickened surface that this costotome is too small to grasp its upper or lower border. The other resected rib has developed a large callus on its end, and is trying to unite at a right angle with the rib below: one can see where the end of the rib was turned in when it was divided at the time of the previous operation. He who should see this pathology to appreciate the extent to which the present procedure must be carried is the physician who first opened the abscess-cavity. The walls of the cavity will never contract without intervention. There is the cavity with the enormous quantity of pus and the leather-bottle walls, with which we have to deal today. It measures eight inches in length by six in width.

Judging from present appearances, there is no doubt that when the physician opened the chest originally he found a large cavity. This operative field is readily sterilized, and sterilization is a very important step. I must excise six ribs in all, beginning with the tenth rib and extending upward, and in performing this Estlander operation I shall employ the subperiosteal method.

There are three types of thoracoplasty—the Schede, the Estlander, and the Delorme. The Schede and Estlander methods have for their purpose the production of collapse of the chest-wall by multiple rib-excision. The Delorme operation involves a decortication of the lung by removing the contracted, leathery, visceral pleura. The ribs are allowed to stay in place, for after decortication the lining expands until it contacts with the chest-wall. The lung does not always remain expanded, however, but often collapses, and the same pathologic condition recurs.

I insert a stitch to serve in anchoring the flap. The usual procedure is to curette these abscess-cavities, but it is not good practice because of its increasing appreciably the danger of absorption of toxins. If you let the patient alone after resecting the ribs granulation-tissue will eventually fill the cavity. As soon as the septic pus has disappeared the walls of the cavity will unite. This operation will be concluded without performing curettement. I insert a stitch in the fibrous wall at its apex to prevent contraction of the flap upward.

RECAPITULATION

Let the record show that all the sponges were removed from the pleural cavity, and that the field is perfectly dry.

Let the record show further that there was an enormous cavity about eight inches in length and six inches in width; that six whole ribs were excised, going away up beneath the scapula, so as to expose completely the cavity according to the method of Estlander; that the thickened costal portion of the pleura was permitted to remain adherent to the intercostal muscles, while the ribs, from the tenth below to and including the fifth above, were removed by subperiosteal incisions (Fig. 229); that there was not so much bleeding, considering the extent of the operation; and that a rubber drainage-tube was inserted.

POSTOPERATIVE COMMENTS

The operation was very extensive, and therefore involved a great deal of trauma. The patient will be given ⅛ grain of heroin immediately, to combat the effects of the trauma.

Of the three operations referred to above, the most desirable is the decortication of Delorme, but in a large percentage of cases his procedure is unsatisfactory. By that method the lung is torn free by removing all the thickened, fibrosed pleura that covers it. Even though the lung be stitched into place at operation, yet after the operation it often collapses and recedes again, so that you have the same condition to contend with. However, the Delorme operation is the most desirable, since the patients have no postoperative deformity of the chest and yet have full restoration of the respiratory function.

Let us review the features of empyema. The first proposition is that empyema in an adult is associated, in a large percentage of cases, with tuberculosis of the lung; proposition No. 2 is that in children the largest percentage of empyema cases is associated with pneumonia; proposition No. 3 is that the chest should be emptied as soon as the presence of pus has been determined. *Ubi pus, ibi evacue,* except in cold abscesses. The protracted presence of pus, whether in the chest or in the skin, stimulates the formation, first, of granulation tissue and later of cicatricial tissue. When there is a collection of pus in the pleura it should be removed at once. The pus proposition is entirely different from what it is when there is an accumulation of serum. If there is serous fluid in the chest and the patient has tuberculosis, compression of the lung by nitrogen gas is indicated; do not remove the fluid, but let it remain compressing the lung and enforcing rest to that extent.

At a discussion of this subject of empyema in Paris, nearly twenty years ago, the question of the utility of the Dieulafoy aspirator as a curative agent was brought up, and it was declared that the instrument had caused more deaths than any that had ever been introduced into the practice of medicine, not because the patient died directly from its use, but because it removed fluid that was a husbanding fluid—a fluid that, by compression, immobilized the lung during the process of repair of the *lung* lesion. As long as that fluid remained serous there was no reason for taking it out. That has been a very hard lesson to learn. We know that in only a small percentage of cases does this fluid ever become purulent, and in a still smaller percentage of cases do you find tubercle bacilli in the fluid in the chest.

This defensive effort of nature serves to compress the tuberculous lung and protect the pleural cavities. Much the same process occurs in the knee when there is a tuberculous focus in the epiphysis that does not communicate with the joint for months and years, but which keeps discharging an effusion into the joint, because the inflammatory condition causes these two surfaces to rub together until they adhere. The pleural fluid contains but a small number of polymorphs—only ten in each $\frac{1}{6}$ objective field.

An empyema in children associated with pneumonia should be evacuated within a week. Wherever pneumonia does not clear up within fourteen or fifteen days there is something "rotten in Denmark." At the expiration of that time the physician should explore the chest with his smallest hypodermic needle and find out what is "rotten in Denmark," and not wait from six days to six months, when well-nigh hopeless pathologic conditions will have developed. There is no trouble about puncturing the pleura if you use the right needle, and but slight danger if the little operation be performed properly. Having determined the presence of fluid, it should be removed *secundum artem.*

I remember an incident that occurred in the practice of my late partner, Dr. Lee. He had a forceful and convincing way of expressing himself. He received a letter from a physician in northern Michigan to the following effect: "Dear Doctor Lee: I have been treating a case of pneumonia for six months and it has not cleared up." Dr. Lee answered, advising him to explore the chest with a needle. In a few days he received this reply from the Michigan physician: "I put in the needle and drew out a gallon of pus. The longer a man lives, the more

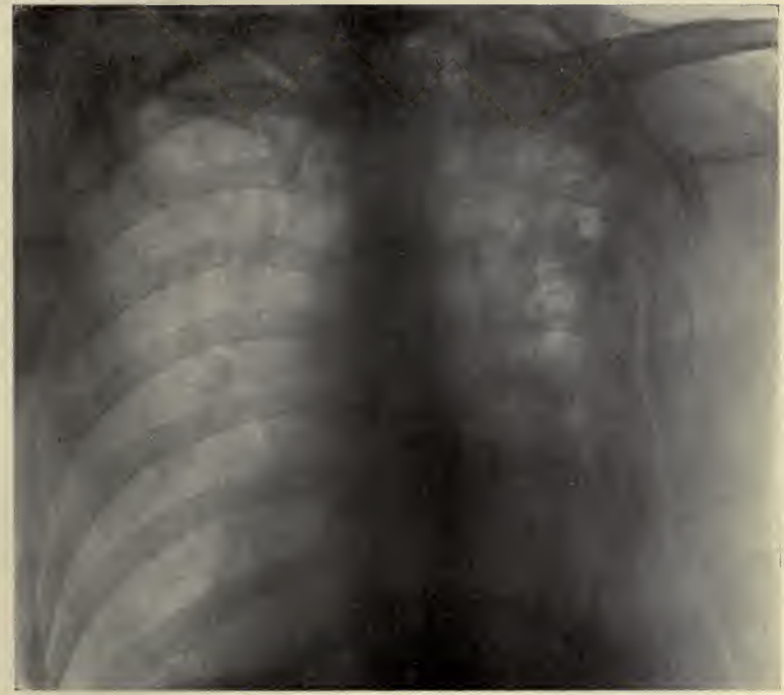


Fig. 229.—Postoperative skiagram of right thorax after resection of ribs by Estlander's method. The vertebral stumps of the resected ribs are clearly shown. Note fibrotic changes that have taken place in lung and pleura (anteroposterior view).

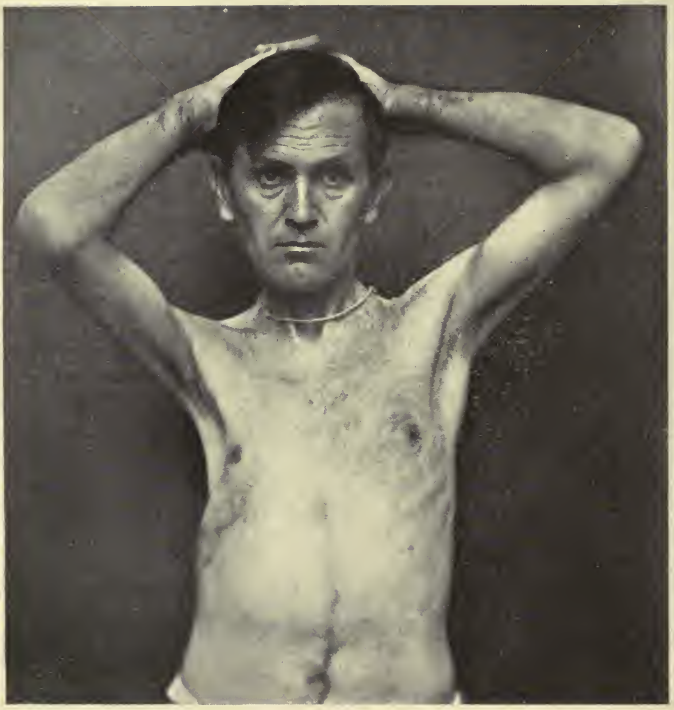


Fig. 230.—Postoperative flattened appearance of right side of chest after Estlander's operation. Sinus completely closed (anterior view).

he learns." I would not like to tell you what Dr. Lee said, but it was something that was in no way complimentary to the Michigan physician.

The greatest cause of mortality in medicine to-day is not cancer, nor tuberculosis, nor acute infections: it is *procrastination.* This statement follows right down through the gamut of diseases. Behold what this poor patient has had to endure unnecessarily! Had the collection of pus been recognized early, it should have been aspirated and formalin injected, and had the fever persisted, reaspirated and reinjected a week or two later, repeating this sequence of treatment until the fluid finally became bloody. When you encounter a huge empyema cavity full of pus the temptation is great to make a huge incision into the chest, draw off the fluid into a basin, and say to the people, like little Jack Horner: "Oh, what a big man am I!" because you have captured such a great prize in the amount of pathologic products. But the burning question is, How are both the breach in the ribs and the pyogenic membrane in the pleural cavity, with their inelastic walls, going to close?

This case illustrates schematically the living pathology and orientation of the wall of a pleural abscess. Note that the thick lower wall of the abscess-cavity can neither expand nor contract: could it contract, the sinus would heal. By operating upon and draining the empyema within the first ten days one guarantees obliterative adhesions of the walls without the excessive thickening of the pleura, resulting in complete resolution and rapid restoration of the patient to his pristine state of health. In postpneumonic empyema in a child it is not necessary to resect a rib, but in the same condition in an adult it is better to do so.

In the Delorme operation the work taxes the patient much more severely because one has to tear off from the lung all the thickened and contracted visceral layer of the pleura—a very annoying procedure before the lung is fixed.

POSTOPERATIVE NOTE

Dr. Murphy (July 9, 1914—six days after operation): There is the extensive concavity which resulted from excision of the ribs. The wound is healing *secundum artem;* but very slight discharge is present. The stitches will not be removed yet: it is too early. We shall dress it with a compress. Since by this time the vitality of the flap is insured, we shall strap the chest with adhesive plaster in order to make a good compression of these surfaces and to produce a coaptation of the granulating pleural surfaces beneath.

[*N. B.*—December 15, 1914, nearly six months after operation. The patient writes that the wound is perfectly healed, that the discharge has stopped, and that he has gained 44 pounds (Fig. 230).]

[Beneficial results of vaccine therapy in four cases of empyema have been published in the CLINICS for August, 1913, p. 551; an illustrated diagnostic talk on empyema, with comments on the treatment, appeared in the CLINICS for April, 1914, pp. 194–206; a case of bronchiectatic cavity treated by nitrogen gas compression was reported in the CLINICS for August, 1915, pp. 705–712.]

PERICHOLECYSTIC AND PERICOLONIC ADHESIONS—RELEASE—OMENTOPLASTY; OBLITERATIVE APPENDICITIS AND PYLOROSPASM—APPENDICECTOMY

Summary: Clinical history of patient; comments—jaundice in cholelithiasis; gall-stone colic may be simulated by gastric ulcer; value of the finger-percussion method; diagnosis of upper abdominal lesions best established during the attack; operation—pylorus bound down and sphincter pylori hypertrophied; extensive pericholecystic adhesions found; adhesions thicken when exposed to traction; colon and gall-bladder bound firmly to liver by a dense adhesion; method of preventing reformation of adhesions; condition of appendiceal zone; removal of appendix; omentum arranged so as to prevent postoperative adhesions; importance of making an ectropion of cut peritoneal edges; recapitulation.

HISTORY

The patient, a male aged forty-six years, was admitted to hospital June 14, 1914. The family and past histories are negative.

About fifteen years ago, in 1899, the patient had severe attacks of colic, with much cutting pain in the upper right quadrant of the abdomen—so severe that they caused him to lie down upon the ground, doubled up with pain. He does not recall any fever, chill, vomiting, or jaundice at the time, but does remember having had much pain on pressure over the region of the gall-bladder, and this tenderness still persists.

The patient states that these attacks lasted until morphin was administered: one attack continued from the middle of the afternoon until the evening—a period of about five hours. He recalls five similar attacks which occurred four weeks apart. He states also that he has often had painless attacks of indigestion, but does not remember their frequency or duration. In these attacks he eructated gas and belched up mouthfuls of sour food. He did not notice any relation between these attacks and eating.

The present trouble began eight years ago—in the fall of 1906. At an indefinite time after eating he noticed a sensation of fulness and of pressure under the left lower costal margin, with eructations of gas and regurgitation of two or three mouthfuls of food. There was never any pain, aside from the distress caused by the gas, the passage of which was followed by relief.

This condition has gradually become worse, and the patient vomits sour material more frequently. There has never been blood in the vomitus, nor in the stools. He says that the stomach was washed out and food found retained six hours after ingestion. The physician states that the patient was given a bismuth meal, and that a skiagram taken after the meal revealed that the pylorus was down almost into the pelvis, and that the stomach was dilated, with its greater curvature in the pelvis.

COMMENTS

Dr. Murphy (June 16, 1914): Does the history state that the patient did not have jaundice, and that he had these attacks of colic many years ago?

Intern: Yes, doctor.

Dr. Murphy: He did not vomit early in the trouble, as is so characteristic of reflex vomiting. Many years ago, in the early stages, he had a history of an attack of cholelithiasis, which was typical except in one detail. Attacks of cholelithiasis are at times not associated with jaundice. At one time we checked up on jaundice as an accompaniment of cholelithiasis, and found that 86 per cent. of the patients with cholelithiasis upon whom we were operating at that time—a period when we were operating upon a great many more gall-bladder cases than we are now—never had jaundice in the early course of their disease, evidence showing that jaundice is one of the late symptoms of cholelithiasis. With this patient the syndrome of cholelithiasis cleared up and has not reappeared.

In addition to the history of gall-bladder disease he has never vomited blood, never had melena, and never hematemesis. Now the pylorus has dropped away down; the lower border of the stomach has ptosed to below the brim of the pelvis, and all the intestines are down in that position. He is still sensitive to perpendicular finger-percussion (Murphy) over the gall-bladder.

The one exception in which colics resembling gall-stone colics occur is in the case of gastric ulcer. In order to make a differential diagnosis—and the diagnosis cannot be made from the history—you must employ the method of perpendicular finger-percussion over the area involved during the attack. I recall my first experience with the finger-percussion method. The patient was a police officer who was laid up every month or two with attacks of colic and severe pain. Each attack lasted two or three days. He came in to see me between the attacks. I could find nothing to indicate anything but gall-stones, the symptoms being pain followed by severe vomiting, but no vomiting of blood. I told him then to come back when in the throes of an attack. One day he came in suffering an attack of pain, and I was able to outline a point of sensitiveness in the midline high up beneath the ensiform, at a distance to the left of the gall-bladder of from two to two and one-half inches. After examining the feces I made a diagnosis of gastric ulcer. Operation revealed a gastric ulcer as large as a silver dollar, but the attacks were typically periodic, like those due to gall-stones. I cite this case to show how diseases involving the pyloric zone of the stomach, the duodenum, and the gall-bladder give rise to similar symptoms, and wish to emphasize the fact that if you see the patient *in* the attack you can make the diagnosis without difficulty.

OPERATION

[Incision.] Now the first thing you note is what? That this colon does not come up: it is fixed to the under surface of the liver by a cicatricial band. Here is the pyloric zone of the stomach; I am bringing the stomach out for examination; you see the lesser curvature. Eighty-four per cent. of all gastric ulcers occur in the pyloric zone and in the region of the lesser curvature (Mayo). The pyloric zone still does not come into the field.

Let the record show that the pyloric sphincter is very much hypertrophied—a condition that signifies some source of irritation which stimulates it to frequent and continued contractions.

We shall elongate this abdominal opening downward in order to obtain a better view. Now there the pathologic record of the gall-bladder shows! In place of its being a free gall-bladder it is entirely buried in adhesions—a record that is written better by pathology than by the history. He had trouble not only within the gall-bladder, but also outside of it. He has a wider extent of pathology because the inflammatory condition extended down below the area of the liver.

The regional structures are united organically to the liver, a union evidently the sequence of an infection that involved first the gall-bladder, since the adhesions all lead directly up to the gall-bladder itself. I do not wonder that he had trouble! I am now pulling the liver up over the costal margin. So far as the health of the patient is concerned these adhesions are worse than Lane's kinks. The solidity of that mass of fibrous tissue indicates the seriousness of the adhesions. Wherever you have an adhesion that is subjected to the effects of traction, it thickens, just as in the case we cited to you yesterday, in which scars extended up over the axilla. Exactly the same proposition holds here.

I have not palpated that gall-bladder for stones yet. There are evidences here of chronic cholecystitis with pericholecystitis. I must endeavor to free the gall-bladder. I am approaching the gall-bladder from behind, letting this firm, anterior adhesion, which if disturbed might tear the gall-bladder, remain. There is the adhesion that has been subjected to traction: it is so strong that I can lift the patient clear off the table with it. See what happens when I free this dense adhesion! The liver comes back to its normal position. The same adhesion caused the angulation of the pylorus.

The thickening of a scar when subjected to traction corresponds to other manifestations of nature. If you want to develop a stunted tibia, how do you do it? By exercise. If you insert a bone-graft to support a portion of the tibia, the graft itself will not grow in circumference until the patient starts walking upon the leg, then the tibia hypertrophies until it is of sufficient size to bear the entire weight of the patient.

Let the record show that the gall-bladder was bound in a mass of firm and resistant cicatricial tissue; that there had been pericholecystitis which caused organic union of the hepatic flexure of the colon to the gall-bladder, and united both the colon and the gall-bladder to the under surface of the liver, and that this band had developed to such extent that the patient could be lifted by it.

The liver remains where it belongs. What will prevent that adhesion from recurring? Nothing: he will have more adhesions unless I excise the gall-bladder. Why should I excise it? The acute inflammatory infection of the gall-bladder has subsided long before this, but in subsiding left these dense adhesions as a legacy. I must provide some means that will prevent that dense adhesion from becoming fixed to the intestines or stomach as it was before. How shall I prevent this? By taking the omentum that was bound in the pelvis, turning it up, and interposing it between the duodenum, stomach, and the intestines on the inner side and the gall-bladder and liver on the outer side. That brings the transverse flexure of the colon out of the pelvis and protects the operative field from the reformation of adhesions.

Let us investigate the condition of the appendiceal zone. There is the terminal coil of ileum. Here is the caput coli. Let me see what is left of the appendix. Here is the appendix bound down and firmly adherent in the pelvis, showing that the patient has had some trouble in the appendiceal zone also. I must free the appendix below and endeavor to bring it up into the field. Now I free the tip of the appendix from the adhesion that binds it down. We shall bring the appendix up into the operative field so that we can remove it. A portion of the organ is the seat of obliterative appendicitis, but there is evidence that the appendix had perforated into a coil of intestine during one of the attacks. This is a bit of pathologic testimony. There is organic cicatricial tissue upon the wall of the intestine. You see the pathology of the appendix, now freed from the intestine. The mucosa has disappeared from most of the appendix, but some mucosa is left over on the side. I am inserting a puckering stitch with which to bury the stump of the appendix. This suture ligates the appendiceal artery. I press back the contents of the unobliterated portion of the lumen and sever the appendix with the electric cautery. Now we ligate the base and bury it in a pouch of the cecal wall. The purse-string suture should suffice to secure the stump, but in addition we insert a straight Lembert seromuscular stitch into the cecum after drawing the purse-string suture taut. We pick up the peritoneum on the one side and then on the opposite side of the base of the appendix and make an overlapping of the wall of the cecum about the site of the purse-string suture. Now we have three obstructions to leakage there, namely, the ligature on the stump of the appendix, the purse-string suture, and the Lembert stitch.

Now we return the cecum and prepare to cover up this exposed area in the gall-bladder zone. This gall-bladder forceps is a safe forceps and merely steadies the gall-bladder without injuring it. I am bringing that omentum up and spreading it out over the under surface of the liver and around the gall-bladder, so that the intestines cannot come over to the liver-area and cannot contract adhesions again.

Let the record show that while there was no ulcer of the stomach, yet there was a very pronounced hypertrophy of the pylorus, which caused us to search for some source of irritation lower down in the alimentary tract; that we found such a source of irritation in the appendix; that the appendix had been a source of recurrent trouble, and was the site of obliterative inflammation.

I shall arrange this omentum so as to get the best results in preventing postoperative adhesions. I tack it above and again below, so that hereafter it will rest in its new position. I shall make another tacking of the omentum to the edge of the liver. The fundus of the gall-bladder has no peritoneal covering, and, if left bare, will become adherent to any structure with which it comes in contact. I am therefore covering the fundus with a portion of omentum to prevent its becoming adherent to the intestines.

In closing the wound we wish to make an ectropion of the cut

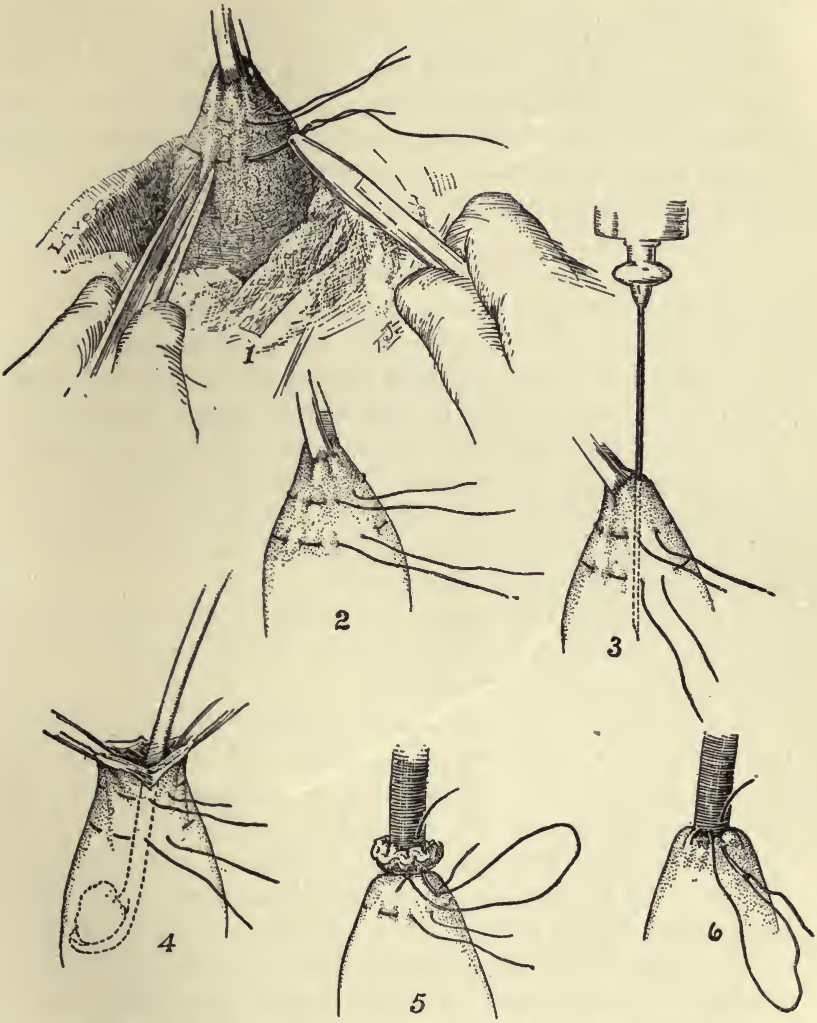


Fig. 231.—A series of illustrations showing the steps of cholecystostomy as performed by Dr. Murphy. Dr. Murphy's technic of cholecystostomy: 1, Shows the method of inserting the circular purse-string sutures into the gall-bladder. 2, Shows the circular sutures *in situ.* 3, The aspirating needle is inserted to withdraw the pus and bile before opening the gall-bladder. 4, The gall-bladder is divided and the Mayo-Robson spoon is inserted, and elevates a calculus. 5, The upper stitch is tied, acting as a hemostatic suture, and is fixed with the same strand of catgut to the tube. 6, Shows the first row of sutures invaginated, and the gall-bladder again fixed to the tube by the same stitch, including only the serosa and muscularis. This insures a permanent entropion of the first row of stitches.

edges of the peritoneum. If we do not turn the peritoneal edges outward, we shall have some of the viscera contracting adhesions to the anterior abdominal wall, and in this situation the adhesions are the most serious of all. Adhesions are apt to be charged with a great many more crimes than they actually commit. This patient had ample pathologic basis for his symptoms.

RECAPITULATION

Let the record show that there was no gastric ulcer; that the gall-bladder was entirely embedded in adhesions and hidden from view; that these adhesions formed a strong band which involved the colon, keeping it on great tension and binding it in behind the liver; that there were no stones in the gall-bladder; that the appendix was exposed and exhibited obliterative inflammation; and that the appendix was bound down in the pelvis and showed evidence that there had been at one time a perforation of the appendix into the intestine.

I do not know when the attack of perforative appendicitis occurred, because there is no clue to it in the history: whether it was very early in life is conjectural.

Let the record show further that in closing the abdomen the operator made a careful inspection of the field to see that there were no sponges left, and that Sister Ludwina reported the sponges and instruments counted and correct.

Let the record show also that the adhesions on the hepatic flexure, which bound it firmly to the under surface of the liver, had developed into a firm cord that was so strong that the patient could be lifted by it; that when this dense cord was cut the tension upon this portion of the intestine was relieved; that after releasing the gall-bladder and exposing and freeing the adhesions, in order to prevent their recurrence a portion of the omentum was sewed over the gall-bladder and over the pyloric end of the stomach, fastening the omentum down into the hepaticoduodenal fossa so as completely to exclude the gall-bladder zone from the intestinal and gastric zones: in addition to preventing recurrence of these binding adhesions the omentum thus employed will aid in holding the transverse colon up; and finally, that the appendix was enucleated, its adhesion to the pelvis separated, and the organ removed in the usual way.

Visiting Doctor: Did the inflammatory trouble around the gall-bladder originate from the appendix?

Dr. Murphy: No. I think that these upper adhesions undoubtedly originated from the gall-bladder itself when he had those gall-bladder attacks and the attacks of pericholecystitis, and that the lower adhesions originated from the appendix.

[Other cases of pericholecystitis have appeared in the Clinics as follows: April, 1913, pp. 243–247 and 249–257; April, 1914, pp. 237–245; and pericholecystitis has been referred to as a cause of adynamic ileus in the Clinics for August, 1914, p. 629.]

TUBERCULOSIS OF FALLOPIAN TUBES—RESECTION; RETROVERSION OF UTERUS—CORRECTION; SUPPURATING DERMOID CYST

Summary: Clinical history of patient; examination; tuberculin test; comments and operation—adhesions encountered; fimbria of oviducts sealed; retroversion of uterus; oviducts excised, stumps buried; correction of retroversion; destiny of the adhesions; raw peritoneal surfaces covered; recapitulation.

HISTORY

The patient, a female, aged twenty-seven years, came to the office on November 2, 1914. The family history revealed that one aunt died of pulmonary tuberculosis fifteen years previously, but that she did not live in the same house with the patient. At the age of thirteen years the patient had typhoid fever and was in bed six weeks. For four years after this disease she was very nervous. Two years ago she was operated upon for appendicitis. The menstrual flow began at fifteen years of age, was regular, and occurred every twenty-eight days, each mensis lasting four or five days and requiring two or three napkins a day; no cramps accompanied the periods. The latest mensis occurred on October 15, 1914.

The present malady began two years ago, in November, 1912, with an attack of dull pain in the lumbar region of the spine. For the next twelve hours she was nauseated and vomited four or five times. There was tenderness on pressure in the right iliac fossa; bending of the back made the pain worse. There was a fever of 101° F. to 103° F. Urinary bladder symptoms were absent. She remained in bed two days. One month later, in December, 1912, a second, similar attack occurred. The third attack took place more than a year after the second—in February, 1914. After the third attack she was operated upon for appendicitis over eight months ago—on February 25, 1914. The pain in the lumbar region of the spine continued.

Shortly after the third operation, on March 15, 1913, she had a chill, and fever of 102° to 103° F., and was delirious two days. The pain in the back became worse and radiated down along the right lower extremity, leading her to think that the hip-joint was involved. She was in bed for over two months—until June 1, 1913. She began to walk for the first time on June 12, 1913, and she walked with a limp. Bending of the back made the pain still worse. Since getting up and about she has continued to have pain in the lumbar portion of the spine and in the right hip.

Eight months ago—on March 15, 1914—the pain in the hip and back became worse. She had a chill and fever, but does not know how much fever; nevertheless, she continued to work. Since the attack of tonsillitis she has had a dull pain in the right hip and has walked with a limp.

Examination by Dr. Murphy on November 2, 1914, showed an area of infiltration around the right hip. A tuberculin test and a leukocyte count were ordered. The spine is to be reëxamined in the near future.

A tuberculin test, 5 minims of dilution No. 2, was given at 7 P. M. on November 2, 1914. The temperature at 6 P. M. on the second was 98.6° F.; at 9 P. M., 99.5°. On the third, at 9 A. M., 99.5°; 12 M., 98.6°; 3 P. M., 99.5°; 6 P. M., 100.5°; 9 P. M., 101.5°. On the fourth it was 99° at 9 A. M. and 99.5° at 12 M.

The patient was reëxamined by Dr. Murphy on November 4, 1914, two days after the first examination. She had been sick for twenty-four hours after the tuberculin test, and had a fever of 102° F., a chill, and general sensations of distress, somewhat resembling the reaction of tuberculosis to the test. Reëxamination of the hip shows abscesses about the trochanter, the origin of which is either trochanteric infection or infection invading the psoas muscle. She also has pelvic infection and retroversion of the uterus.

Vaginal examination made by Dr. Murphy on November 11, 1914, at the hospital shows great degree of sensitiveness in both fornices, but no indication of a mass. There is also retroversion of the uterus.

OPERATION

DR. MURPHY (November 18, 1914): [Incision.] You will note that in making the division of the abdominal wall I first incise the sheath of the rectus muscle one-half to three-fourths of an inch to the left of the midline, and then displace the rectus muscle. I am also exposing the peritoneum one-half to three-fourths of an inch to the left of the midline before dividing it. We do not know what we are going to find when we get inside. We know that there are adhesions. The history revealed an operation for appendicitis, which explains that history of trouble in the pelvis. Upon entering the peritoneal cavity I find, remote from the previous position of the appendix, organic adhesions to the anterior abdominal wall. The intestines are all matted together and greatly displaced. We now have the intestines freed for the first time as I lift them up from the mass of adhesions. Are these adhesions the sequence of a peritonitis resulting from the previous operation, or are they the sequence of a peritonitis from an old infection, which sequence is common in the type of disease that corresponds to her clinical history?

You will note as I work my way into the pelvis that I am trying to separate the fixed intestine from the anterior abdominal wall and to keep away from the bladder. Now let us see how extensive these adhesions over on the right side are. You see the cecum is surrounded by adhesions which look like postoperative adhesions following appendicectomy, but of that we are not sure. The small intestine is matted together as though the result of tuberculosis, or even of an extensive non-tuberculous peritonitis. See those cobweb adhesions that are so common in peritoneal tuberculous processes! Let us see what they show. What I would like to do is expose the Fallopian tube. She had this operation for appendicitis nine months ago—in February, 1914. We shall enlarge the opening through the abdominal wall.

Here is the sigmoid loop running across the brim of the pelvis. I am going to see if I can make a flank movement upon the sigmoid and get down into the pelvis: I am trying to get into this line of trenches and come around; now I have succeeded fairly well in getting into the pelvis. Remember that we are separating these adhesions not for the mere purpose of separating them—that were a futile procedure. I am separating them for the purpose of getting into the pelvis. There is the urinary bladder below the intestine. We want to find the cause of the adhesions and, if possible, eliminate it.

Now we put in the Mayo-Simpson retractor. From the way these viscera are involved it looks as though the upper adhesions at least were due to the chronic peritonitis that developed after the operation for appendicitis—a condition that is lamentable but unavoidable. In some of these patients a chronic type of peritonitis sets in that resembles very much the keloid condition following excision of diseased lymph-nodes of the neck, and occasionally following vaccination.

Now, as we displace all the viscera out of the pelvis the real condition looms up as an encapsulated tuberculosis, with the uterus pressed down into Douglas's pouch. Let me show you this tuberculous abscess. These adhesions are the right type of adhesions for the disease present. It looks to me as though she had also a pus-tube in association with the tuberculous abscess, but the latter is the condition that is in consonance with the clinical history—tuberculosis. I shall deliver the tubes and see whether the tubes are tuberculous. There is the tube on the one side, and here is the tube on the other side.

Let the record show that the fimbriated ends of both tubes are completely sealed, showing that if the original infection was Neisserian or tuberculous, it had closed subsequently both fimbriated ends.

I am determining the orientation of the tuberculous abscess to the tubes. The whole picture is one of tuberculosis. The fimbriated ends of both tubes are apparently destroyed. There is the ovary on the left side, and here the abscess, which extends over to the fimbriated end of the tube on the opposite side. This is a pretty picture because it shows both fimbriated ends becoming closed from the proximity of one centrally situated abscess.

On the left side the abscess is attached to the ovary. There is the fimbriated end of the right tube also involved in an abscess. That is the round ligament, which is independent of the tube—I shall keep it for the treatment of the retroverted uterus. I must separate the uterus entirely from the tube.

INTERN: I have the report on the tuberculin test. The patient had a chill, and a fever of 102° F.

DR. MURPHY: She gave a positive response—a fever of 102° F. with a chill. That settles the question of tuberculosis, and corresponds exactly with my findings. Everything is perfectly clear; the clinical history is right, and corresponds with the physical findings.

This tube is of no use as a tube any longer because its function is irreparably destroyed. I am freeing the tuberculous abscess from the ovary and the tube on the left side, but not on the right. Both the abscess and the tube seem to have coalesced. I am now spreading out the remnant of the tube so as to get rid of this focus of inflammation.

As the history shows, I did not feel these pelvic masses when I made the examination: I could feel only the retroversion, but the history of recurrent chills and fever told the story of other troubles. I am freeing the round ligament from the mesosalpinx. After removal of the tube I am cauterizing the stump with 95 per cent. phenol, and then I bury it beneath the broad ligament. From our interpretation of the lesion this is not a case of venereal infection—it is one of tuberculous infection. I wish to conserve both ovaries: this woman is comparatively young and we can save both ovaries without increasing the hazard to the patient, because the ovary is not the cause of this condition. I shall not cover that raw surface until after removing the other tube.

Now I want to show you the right tube. See it! There is the right ovary. There is the most recent Graafian follicle to rupture. [The patient finished menstruating just before the operation.] I shall free this attachment of the ovary to the tube. The right tube is irreparably damaged. I shall ligate the tube, divide it with the cautery, and bury the stump behind the peritoneum on this side also, and after that shall shorten the round ligament and cover the whole raw surface. In almost the same way as an appendiceal stump, that tubal stump will be turned in behind the peritoneum, so that it will not be free to discharge any more pus or tuberculous material into the peritoneal cavity. All raw surfaces are covered in. The tubal element is out of commission.

Now I wish to correct the retroversion of the uterus. You see there the round ligament, and here is the vulsella holding the uterus up. I am going back of the intertubal line. We do not have to reckon with the tubes here any longer. I am going to take up the margin of that ovary, the margin of that lacerated surface, and the round ligament, and throw the whole tissue over upon the posterior surface of the uterus to sling the uterus forward. See where the round ligament comes now! In place of coming over to the anterior surface of the uterus it is attached permanently to the posterior surface of the cornu of the uterus. We know, much to our sorrow, how well the uterus stays in its new position. A fortnight ago we had to excise a fibroid in a woman upon whom this operation had been performed twelve years previously, and it was the hardest removal of a fibroid that I have ever had. After operation the patient got along without an unpleasant symptom. Now we bring up the other round ligament and fasten it also behind the cornu on its own side of the uterus.

What will become of all these adhesions? The great bulk of them will disappear entirely. Why? Because they are of tuberculous origin—take out the focus of infection in tuberculosis and the rule is that the adhesions disappear. You would be surprised if I reoperated after six or eight months without finding any traces of adhesions, and that they had all disappeared with the exception of a few organic ones. I am going to sew the peritoneum across the tissues we have just been suturing, to avoid additional adhesions to the scar. There is the urinary bladder in the field, normal and contracted.

That was a gratifying case, because it cleared up so nicely at operation. You saw the adhesions on that posterior peritoneum: they extended all the way over to the sigmoid from the position where the appendix had been removed. I shall turn in this sigmoid upon the raw surface of peritoneum in order to prevent adhesions to the anterior abdominal wall.

RECAPITULATION

Let the record show that upon opening the abdomen we found firm adhesions to the anterior abdominal wall; that upon freeing these adhesions we encountered typical cobweb fixations over a great extent of the small intestines; that we succeeded slowly in freeing the intestine and entering the free peritoneal cavity, coming to a position where the adhesions were not so extensive; that then we came down upon an encapsulated tuberculoma, whose capsule was formed by a union of the fimbriated ends of both tubes; that both tubes were tuberculous; that this abscess was encapsulated and free from anything but light adhesions; that the peritoneum and intestine were then freed so as to expose the pelvis; that the uterus was elevated from its retroverted position, exposing the tubes, which were enlarged only at one area back of the fimbriated end; that both tubes were then removed, the stumps ligated, cauterized with 95 per cent. phenol, and then buried beneath the peritoneum of the broad ligaments; and that finally the round ligaments were sutured to the fundus of the uterus posteriorly, in accordance with our usual operation for the correction of retroversion.

You can see now that all the bleeding area was made extraperitoneal. The raw surface is covered over by peritoneum and cannot contract adhesions. I have never seen a case of that kind before: the clinical history is so superlatively in consonance with the pathologic findings.

Let the record show that the tuberculin test made in the office showed a reaction—fever of 102° F.; that the patient had a chill, headache, malaise, and was uncomfortably sick—the reaction showing a superlative degree of tuberculosis and showing it in the active stage; and finally that all these findings corresponded with the clinical course of the disease.

Let the record show that Sister Victorine reported the sponges counted and correct, and that the operator inspected the field on closure to see that no sponges or instruments were permitted to remain in the peritoneal cavity.

Let the record show also that after fixing the uterus the sigmoid was turned over upon the fundus of the uterus to cover the abraded pelvic area and to prevent the formation of organic adhesions on the fundus of the uterus; that we predict that the cobweb adhesions which existed throughout the lower abdomen will disappear within a short time; and that we did not expose the cecal area from which the appendix had been removed.

You can see from the story that an inspection of the pelvis had not been made at the time the appendix was removed, or the situation could have been cleared up right then and there. The severity of the symptoms forced us to go in again such a short time (eight months) after the original operation.

Pathology.—Both the tubes were tuberculous. The central abscess was due to a suppurating dermoid cyst. There was a mixed infection in each of the tubes, which accounted for the closure of the fimbriated ends and probably accounted also for the infection of the dermoid cyst. The abdomen was closed without drain. Complete primary union resulted.

SARCOMA OF OVARY—ABLATION

Summary: Clinical history of patient; comments—fibroma *vs.* fibrosis; fatality of cancer of cervix; operation—ablation of malignant tumor of ovary; citation of illustrative cases—essential hemorrhage or local hemophilia; recapitulation; postoperative comments—sarcoma of kidney, ovary, or testis liable to be bilateral—carcinoma not liable; site of early metastases from sarcomatous testis; sarcoma of left testis metastasizes through lymphatics.

HISTORY

THE patient, a female aged fifty-eight years, was admitted to hospital June 14, 1914. The family history reveals that one brother died of tuberculosis forty years ago. The past history shows that the patient had malaria thirty years ago. Menstruation began at fourteen years of age, was of the twenty-eight-day type, regular, and was painful during adolescence only. She has been married twenty-nine years and has two children. Thirteen years ago she had one miscarriage at the third month of pregnancy. She has never passed clots at menstruation.

The patient states that following the miscarriage of thirteen years previously she noticed a decrease in menstruation after the age of forty-five, the flow gradually diminishing in amount, necessitating but one napkin a day, and also that it was shorter in duration. After these effects of the miscarriage had adjusted themselves she menstruated regularly again up until fifty years of age, when she began to miss one or two months at a time. At the age of fifty years she menstruated every three weeks, each mensis being of the usual amount and requiring two napkins a day for four or five days. There was no pain, no discharge, no odor, and there were no blood-clots. At the age of fifty-three years the menopause was terminated and she noticed no further flow until two months previous to admission,—on April 9, 1914,—when there was a faint trace of blood for two or three days—barely enough to soil a towel when bathing. Two weeks later she noticed the same faint trace again for two or three days. She did not use a napkin this time. Six weeks before admission, on May 5th, there was the same stain on a towel for the second time. Nine days ago, on June 5th, there was another show (the third), which lasted two or three days. On the night before admission, on June 13th, she flowed a little more. She says there was a stain on her night-dress, and that is all she has noticed. There has been no pain, no loss of weight, nor odor to the discharge at any time.

COMMENTS

Dr. Murphy (June 15, 1914): Now this cervix does not look as though it had anything the matter with it, still the flowing has been just enough to make us suspicious that she has trouble with the epithelial cells that line the uterus higher up. If she had malignant disease, that cervix on curettage would come out with spoonsful of necrotic tissue: on the contrary, the cervix is firm, hard, and resistant, and in no way diseased. She has a retroverted uterus, with either a fibroma or fibrosis, the chances of both being the same as far as the flowing is concerned, but in this case I should say it is a fibroma instead of fibrosis. Remember that patients have the same type of bleeding with fibrosis that they have with a fibroma.

Let me show you these statistics, because they seem just a little too good to be true (p. 917). If she had a carcinoma that has passed beyond the stage of a small heaping-up of the external epithelial cells within the cervix, that is, an epithelioma, I would estimate her chances of complete recovery as 20 per cent. with this mass involving the os internum, which is the rich lymphatic zone of the cervix, not only of the uterus, but of other organs, as the gall-bladder and the stomach (pylorus). When carcinoma begins in that rich lymphatic zone, early and wicked metastases take place. When carcinoma involves the fundus of the uterus, which, according to the statistics, happened only 18 times in 677 cases, showing how rare it is, there is always *late* metastasis. Why? Because the fundus uteri has such a feeble lymphatic circulation. The lymphatic supply of the fundus of the uterus is just as feeble as it is in the fundus of the gall-bladder. When infection lodges in the fundus of the gall-bladder, there is very rarely elevation of temperature, but when the infection occurs in the cervix of the gall-bladder, there arise chills and fever of severe intensity. I have cited a case thousands of times as illustrative of the great severity of symptoms that can be present with such a small quantity of pus and such a small gall-bladder.

When the cancer is of the squamous type on the outer side, the prognosis is better; when of the columnar type, on the inner side, near the junction of the cervix with the body of the uterus, an unfavorable prognosis always exists. You see the statistics with the new Wertheim operation. In looking over the recent statistics of a surgeon who reported separately a series of general abdominal operations for various conditions and another series of vaginal hysterectomy for carcinoma of the uterus, I found a mortality of 2.5 per cent. in the vaginal series and of 25 per cent. in the abdominal series of Wertheim. An operation that eventuates in a 25 per cent. mortality—one patient in four dying—is a colossal undertaking, no matter whether it be performed for cancer or any other condition. I think the best result that you may expect in cases of cancer of the cervix uteri is permanent cure for one patient in every five. That is a pretty bad proposition.

UTERINE CARCINOMA

Schauta's operation: Vaginal, extensive removal:
    258 cases—mortality not given.
Bumm's clinic: Sigwart reports:
    218 cases—mortality 25.6 per cent.
Kinoshita reports:
    408 operable cases—
        62 palliative (11.2 per cent.).
        207 radical abdominal (31.3 per cent.).
        39 radical vaginal (25 per cent.).
Schottländer and Kermanner analyzed:
    677 cervical carcinoma and 18 uterine from the late von Rostock.

324 cases were operable—256 by abdominal route (18 per cent.), 66 by vaginal route.
The bladder was injured 38 times.
The ureter was injured 16 times.
The rectum was injured 5 times.
Secondary necrosis giving vesical fistula 16 times.
Secondary necrosis giving ureteral fistula 18 times.
Absolute recoveries in 196 cases traced were 36.2 per cent.

Most recurrences were in the vaginal scar; only 4 recurred in the lymph-nodes, notwithstanding that in 23 out of 48 cases in which the nodes were examined cancer was present in the lymph-nodes, which would lead one to believe that many nodes which are carcinomatous heal after removal of the primary tumor.

OPERATION

[Incision.] Now I am lifting the uterus out of Douglas's pouch and swing it up into the field. Here is a tumor resting behind the uterus: it is either a carcinoma or a sarcoma of the ovary. I shall wipe out all that raw area to keep from getting an implantation of malignant cells in the bed of the tumor. We now have the whole tumor out. [Crushes mesovarium with angiotribe.] I shall tie that ligature into the crease made by the angiotribe, so as to obtain direct compression of the vessels. I shall do the same crushing and ligating on the opposite side. We insert a second ligature behind the first. Now we shall section the tumor of the ovary to determine the cause of the bleeding. We had a young woman about twenty-six years of age on the North Side of Chicago who had an essential uterine hemorrhage. The masses were not larger than hen's eggs.

Here is the enlarged uterus, larger than it should be in a woman of her age—past the menopause: we shall not have to remove it because of the presence of the malignant tumor in the ovary. Thus we avoid hysterectomy—a very great saving in a woman of her age with an enfeebled cardiovascular condition.

The radial arteries are unequal on the two sides. The right radial artery is very small, while the left is of about the usual size.

I recall a very trying operation that I performed in which this inequality in the size of the radial arteries was present. I was operating upon a late Secretary of the Navy. The internists in consultation with me were Dr. William Osler and Dr. Jennings of Detroit. Dr. Osler had his finger upon the radial artery on the side where it was small, and Dr. Jennings on the opposite side. I was operating upon the patient under very unfavorable circumstances. Dr. Osler kept repeating every few minutes: "Hurry up, hurry up; he is getting low!" Dr. Jennings, whose finger was upon the larger artery, replied: "No, Dr. Osler, that pulse is not low." You can imagine the position I was in, trying to get out a little infected gall-bladder with no stones in it, but embedded in a mass of adhesions. After the operation I went back into the patient's room and felt the pulse on the side that Dr. Osler had been in charge of, and found it very feeble. Fortunately, I changed sides and felt the pulse that Dr. Jennings had been feeling, and it was perfectly good. The patient made an uneventful recovery.

We have ablated that tumor completely: you see, here is the stump. I wish to cover in that stump so that nothing will become adherent to it.

What is the prognosis now? Good, notwithstanding that in my estimation the tumor is malignant. I think it is a sarcoma rather than a carcinoma, although I am not certain. I operated upon a woman for sarcoma of the ovary who lived six years after operation without any evil results of the neoplasm appearing again.

Let the record show that the other ovary is atrophied to a small, fibrous mass.

I started to tell you about that case of uterine hemorrhage. The patient was a young woman who had borne five or six children within a period of six or seven years. She suddenly developed pain in the abdomen. It was time for her regular menstrual period and she began to bleed very profusely—so profusely that the physician had to pack the uterus. Her baby was six months old. She did not believe that she was pregnant again, and neither did we. Two weeks after the hemorrhage began we examined her carefully and found her very anemic. A transfusion from her brother as donor restored the hemoglobin content. However, she began to lose blood again, but very little, comparatively, and continuance of the hemorrhage with hemolysis caused her death five weeks after the onset of the primary hemorrhage. Autopsy revealed neoplasms in both ovaries, which resembled closely the neoplasm in this patient. The question between the endotheliomatous, or sarcomatous nature of the neoplasm was never decided by the pathologist. The surface of the uterus was just as smooth as the palm of my hand. It was an essential uterine hemorrhage, apparently caused by the neoplasms in the ovaries. We do not know the mechanism by which essential uterine hemorrhages arise, just as we do not know that of essential renal hemorrhages. On microscopic examination there is nothing at all found in the uterus, nor in the kidney in explanation of the essential hemorrhages.

After I came back from Berlin in 1890 I cited a case which Senator diagnosed as essential renal hemorrhage. After removal of the kidney the microscopic examination revealed nothing at all to explain the bleeding, but the patient was exsanguinated to the extreme degree. It was Senator's belief that it was a case of local hemophilia. Local hemophilia is merely another name for essential hemorrhage, and implies that there can be local hemophilia of an organ apart from constitutional hemophilia.

We had another patient with hemorrhage of the same type. One Sunday morning I was called to see the patient during the absence of the late Dr. Henrotin from the city. She was so exsanguinated from the uterine hemorrhages that we did not give her an anesthetic. We performed the clamp operation of hysterectomy. She had borne nine children in rapid succession, and each morning on my way to the hospital I saw those nine children, six of whom were boys, and thought of the colossal responsibility of operating upon the mother of nine children. She made a good recovery. There was no evidence of a lesion of the ovary to explain the cause of the essential hemorrhage, unless the rapidity of childbearing—nine children in ten or eleven years—played some etiologic rôle. When the uterus was removed, it was found perfectly smooth; the microscope revealed nothing. She lived for many years after the operation.

Let the record show that Sister Victorine reported the sponges counted and correct, and that on closing the abdomen we carefully inspected the field to see that no instruments or sponges remained.

RECAPITULATION

Let the record show further that on vaginal examination the cervix was found normally hard and resistant, and showed no evidence of intrauterine disease—findings which were exactly in consonance with the pathologic conditions and with our interpretation; that upon opening the abdomen we found the uterus retroverted and resting upon a hard tumor, which proved to be a solid tumor of the ovary; that this tumor bled when disturbed and suggested either a carcinoma, or a sarcoma; that the uterus was enlarged, but showed no bulging, the bulging from behind being the mass pressing upon the uterus; that we therefore believed that the uterine hemorrhage was the sequence of the neoplasm in the ovary; that we did not perform hysterectomy; that we did not let the abdominal viscera come in contact with the neoplasm during its removal; that we did not have to deliver the abdominal viscera from the wound during the operation; and finally, that there was no enlargement of the right ovary—on the contrary, it was atrophied.

POSTOPERATIVE COMMENTS

The case I cited of the woman who lived six years after the removal of a sarcomatous ovary was the only case of its kind that I have ever seen that did not recur in the ovary of the opposite side. Whenever sarcoma involves the ovary, the testis, or the kidney there is a tendency to recurrence upon the opposite side, and many times both sides are involved synchronously. Of the sarcomata of the kidney which I have met with I have seen none that did not recur upon the opposite side. Some years ago, in looking over Morris's statistics, I found few, if any, authentic cases of removal of the kidney for sarcoma that did not have recurrence within a short time—within two and one-half years. In the ovary sarcomata recur upon the opposite side frequently, though not always—not so uniformly as in the kidney, but still more frequently than in the testis. You see, therefore, that the incidence of recurrence of sarcoma in the three organs upon the opposite side decreases in frequency from the kidney downward. Because of the small, atrophied ovary on the opposite side in our patient we venture to hope that there will be no recurrence.

This liability of sarcoma to involve both sides does not obtain in carcinoma. Any number of cases of carcinoma of the kidney, of the ovary, and of the testis remain perfectly well after operation. We do not know the paths by which sarcoma spreads to the opposite organ. When a sarcoma appears in the *left* testis, by the time the patient comes to the surgeon for treatment you will find almost invariably a metastasis of the sarcoma from the left testis into the left renal lymph-node, which is situated retroperitoneally upon the left renal artery or vein. The lesson to be learned is that, when dealing with a sarcoma of the left testis, it is wise to open the abdomen at once and, exposing the left renal artery and vein, see whether that lymph-node is involved. Sarcoma of the left testis affords one example where sarcoma elects to metastasize through the lymphatics. You remember that in all other sites involved in sarcoma, even in the right testis, sarcoma elects to reach its metastatic position most commonly by the blood-channels. Occasionally sarcoma of the right testis, as well, metastasizes to the renal lymph-node, but less frequently than in the case of the left testis. Carcinoma, as a rule, metastasizes through the lymphatic channels, although there are exceptions to this rule even, as when carcinoma of the thyroid gland travels through the blood-stream to the lungs and other regions. Carcinoma of the thyroid and all carcinomata above the clavicular level metastasize most commonly in the lung and pleura, while all carcinomata below the clavicular level metastasize most commonly in the liver.

[A case of sarcoma of the ovary with rotation of the pedicle was published in the CLINICS for June, 1914, pp. 599–609; sarcoma of the testis is referred to in the CLINICS for February, 1914, p. 72; and June, 1915, p. 485; a case of sarcoma (hypernephroma) of the kidney, with a colored illustration and a table, is described in the CLINICS for June, 1915, pp. 535–548.]

PYONEPHROSIS—INCISION AND DRAINAGE —SUBSEQUENT NEPHRECTOMY

Summary: Clinical history of patient; comments—path by which kidney was infected; operation—incision of left kidney and drainage; *B. coli* infection; second operation—nephrectomy by clamp method; recapitulation; postoperative comments.

HISTORY

The patient, a married woman aged fifty-one years, was admitted to hospital June 5, 1914. The family history is negative. The past history reveals an attack of pneumonia eight years ago—in 1907. Menstruation began at eleven years of age, was regular, of the twenty-eight-day type, profuse—requiring from 15 to 20 napkins a day—and lasted usually from ten to twelve days. She passed the menopause five years ago—in 1909—after an operation the nature of which she does not know.

The present trouble began three or four years ago, in 1910 or 1911, that is, a year or two after the operation. It was the appearance of pus and clots of blood in the urine at this time which first called her attention to the fact that something was wrong in the urinary tract.

She states that immediately after the operation she suffered from retention of urine and was catheterized several days—as often as five or six times a day. After she had recovered from the operation, however, she had no more urinary trouble until a year or two later, when she noticed pus and blood in the urine. At about this time she also had frequent desire to urinate, sometimes as frequently as every few minutes, but she was able to pass only a small quantity at a time, and used a catheter frequently to remove urine that seemed to be left in the bladder. There was no acute pain in the region of the bladder at this time, but aching pain was referred to a point a little below the left kidney region, and more anteriorly than posteriorly. The patient has forgotten the exact duration of this attack. Her condition soon improved, and, with the exception of the occasional passage of blood-clots with the urine, she noticed nothing of moment until about a year ago, that is, in 1913, when the pain in the left side again became severe. Again she suffered with greatly increased frequency of urination, and sometimes had to arise as often as 15 times in a night to urinate, and then passed only a few drops at a time. One year ago she discontinued the use of the catheter because it caused so much pain in the bladder.

On the last Saturday in August, 1913, nearly a year ago, while shopping, she had a severe chill which lasted almost all day. She went home and got into bed, but remained in bed only a few hours, although suffering with severe pain in the region of the left kidney. She was nauseated and vomited almost continually for two days after that, and probably had fever.

After this attack her condition remained about the same, without any notable improvement, and so she came to hospital about three months ago—on March 10, 1914—for further examination and treatment. She remained ten days or two weeks. Since this period of hospital treatment she has not had so much pain nor does she have to urinate so frequently as before.

On June 5, 1914, she again returned for cystoscopic examination. This examination showed the bladder enormously dilated. The left ureter was seen discharging almost pure pus. The urine from the right ureteral orifice also was not entirely clear, but it appeared to contain much less pus than the left.

She was advised to remain in hospital and have the pus-sac in the left loin incised and drained.

COMMENTS

Dr. Murphy (June 6, 1914): We do not intend to excise this left kidney, but merely to drain it. We know that the right kidney is discharging pus with its urine, and we therefore feel that we should save all the renal parenchyma possible.

The patient has had very severe systemic reactions at times with her trouble, accompanied by chills and high fever, but no metastatic abscesses, or joint-infections. The tonus of the bladder detrusor also has been lost in the course of years, so that the bladder is now permanently dilated and has a capacity of about a gallon. What the patient urinates is chiefly the product of overflow from incontinence of retention, and that has probably been the case for years.

My opinion is that the infection followed the repeated catheterizations which were performed to relieve the postoperative urinary retention several years ago, of which the history tells us. As the result of the repeated catheterizations, ascending infection of the urinary tract has taken place,—a cystopyelonephritis,—and during its course urinary retention developed and produced pyonephrosis and probably nearly complete destruction of the renal parenchyma on the left side. By our operation to-day we aim merely to open and drain the pus-sac, which undoubtedly constitutes about all that remains of the left kidney.

OPERATION

[Usual lumbar incision made.] The plan which we are using in the management of this case is one originally advocated by Morris. It consists of a muscle-splitting incision down to the capsule of the kidney, thus opening up to infection as little raw surface as is possible.

Here is the fatty capsule, and just within it the enlarged kidney. There is a large vein running across the surface of the kidney, and I am taking care to avoid cutting it. I am incising the kidney along its convexity: one need not be particular to strike Hyrtl's avascular area exactly in such a sac-like kidney. Here comes the pus! Now we insert this large rubber tube for drainage and are already through with our procedure except the closure of the wound.

Colon bacilli have been present in this patient's urine ever since she has been under observation. Although this infected urine is flowing out over the surface of the wound, the chances are that the patient will secure a primary union of the wound down to the drain. On the other hand, suppuration occasionally develops after an apparently aseptic operation, though we do not know why. At any rate, there are many facts about the circumstances surrounding the formation of pus with which we surgeons are not yet acquainted. After operation for suppurative appendicitis the wound closes by primary intention, probably in eight out of ten cases.

As soon as this kidney is draining properly the bladder irritation will subside. As soon as we see by a functional test that the other kidney is doing all the work of urinary secretion, we shall reopen this wound and enucleate what remains of the pyonephrotic sac. If we should take that step now we might be removing just that small balance of secreting renal parenchyma which is necessary to preserve life.

SECOND OPERATION

Dr. Murphy (July 6, 1914): What did the examination of the urine show after the first operation?

Intern: On the seventh of June, the day after operation, a trace of albumin was present. That was all.

Dr. Murphy: How about pus?

Intern: There was no pus noted at that time. On the eighth of June there was no albumin present. There were from 10 to 15 white cells to the field of a 1/6 dry objective. On June ninth it was noted that albumin was present, and that the microscopic field was packed with white blood-cells. On June tenth albumin was again absent, and there were only about four white cells to the field of a 1/6 objective.

Dr. Murphy: I want to call your attention to that point. As soon as the kidney was thoroughly drained, and so long as it continued to drain adequately, the patient stopped having pus in the urine, because the pus was discharged, together with a small amount of urine, through the lumbar incision. After our drainage operation the patient's temperature remained about normal until the first or second day of July—nearly one month after operation. Then it shot suddenly up to 104° F., and on the next day to 105° F. We know that the right kidney is secreting an adequate amount of fairly normal urine because we have made the "phthalein" test. Therefore, we think it high time that this badly infected left kidney, which is continually threatening to produce constitutional sepsis, should be removed completely, and that is our task to-day. The drainage-tube has been removed, and it is because of the insufficient drainage following its removal that there has been retention of pus and the development of this high and threatening temperature. This kidney is large, surrounded by inflammatory adhesions, and is not going to be easy to remove. We must take extra precautions in our dissection and in our hemostasis in this case because we know that the operation is going to be followed by suppuration within the cavity. My incision for this operation must be much larger than that of the previous operation, because now I must excise the kidney instead of merely opening it, and it is a very large organ.

[Dr. Murphy approached the kidney by cutting through the scar of the previous operation-wound and then enlarging the incision downward and forward around the costal margin.]

I am down upon the kidney now and am endeavoring to dissect around it while keeping inside of its fatty capsule. The landmarks here are disturbed not only by the scar-tissue resulting from my previous operation, but also by the old inflammatory adhesions produced by the long-standing suppuration in this kidney. . . We are down to tissue which we are able to recognize as kidney-parenchyma. We know how close the intestines—especially the colon—often lie to a suppurating kidney, and how adherent they may be. This looks like intestine right here in front of the kidney, shining through the posterior layer of the parietal peritoneum, and it *is* intestine. At last we have a landmark, and, even if it be a sign of danger, we welcome it. This entire field of operation is so infiltrated with the products of inflammation that I believe the only safe measure by which to prevent hemorrhage is to use the permanent clamps on the kidney pedicle here. Now, at last, I have been able to deliver the kidney to the surface and here I have it lying across my fingers. There is the artery. I am ligating it doubly. There is the renal vein; I shall catch it with the clamp, and that will be sufficient. The clamp is the safest hemostatic measure when one has to deal with large vessels embedded in inflammatory tissues, such as are present here. We have the kidney lying entirely outside of these two forceps. We are cutting its pedicle distal to the forceps. The stump is now well cared for. There is still a little bleeding from the connective tissue in the vicinity, but that will soon stop.

RECAPITULATION

Let the record show that we clamped the renal pedicle distal to the ligature on the renal artery. Let the record show also that we excised the kidney without soiling the field of operation; that we lost very little blood, notwithstanding the numerous adhesions that were present; that at the completion of the work hemostasis was perfect; that we included the vein in the clamp because it was situated in a mass of cicatricial tissue on which we did not wish to risk a ligature for fear of its cutting through the infiltrated tissue; that the ureter was very much thickened, and was clamped with the other tissues of the renal pedicle; that the lumen of the ureter was cauterized distal to the clamp with 95 per cent. phenol, so as to sterilize it; that all bleeding points were ligated, and that both the angiotribes were placed with their tips pointing forward, so that they can be easily removed by a posterior displacement of the tips. Let the record show further that two strips of nosophen gauze were inserted for drainage; that Sister Ludwina reported the sponges counted and correct; and finally that the operator examined carefully the operative field for sponges and instruments before completing its closure, but that none was found.

POSTOPERATIVE COMMENTS

We are even a little more careful in making certain that no sponges or instruments are left behind in an operation in this location than we are in intra-abdominal operations. The chances of leaving a foreign body in this locality are, I think, even greater than in the abdominal cavity.

These two clamps are being tied with separate strands of gauze to prevent their becoming accidentally unlocked. We always secure them in this manner. As the blood drains from the wound, the gauze is soaked and is not liable then to become loosened, and so the clamps are perfectly safe as hemostats, being thus reinforced doubly against accidental unlocking. They will be left in place for seventy-two hours: by that time thrombosis will have occurred in the artery and in the vein.

Pathology.—Examination of the excised kidney showed it to be considerably enlarged and to consist of a thin shell of parenchyma surrounding the greatly enlarged and dilated pelvis, which contained pus. The pelvic mucosa was greatly thickened. Besides being narrowed, the cortex was much increased in consistence because of the extensive formation of scar-tissue within it. Scattered throughout the cortex were numerous small areas of necrosis and many tiny pus-foci running out from the pelvis more or less in streaks, thus indicating that the process is an ascending infection—a pyelonephritis. There were no abscesses in the cortex resembling metastatic hematogenous abscesses.

[*N. B.*—The patient made an uneventful recovery.]

[A case of pyonephrosis, together with a table of tumors of the kidney, was published in the CLINICS for December, 1913, pp. 967–977.]

URETERAL CALCULUS — URETEROTOMY — REMOVAL OF CALCULUS

Summary: Clinical history of patient; comments — appendicitis *vs.* ureteral calculus; table showing analysis of cases of urinary calculus; diagram showing frequency and location of impacted urinary calculi; albumin and blood in urine after bimanual palpation of kidney; palpation of a kidney the seat of a tumor thus of diagnostic aid (artificial hematuria); causes of "idiopathic" hematuria; diagnosis of renal calculus—value of "fist-percussion" test in acute obstruction of ureter and in renal infarcts; value of skiagrams in detecting ureteral calculi; means of detecting the location of urinary calculi at operation; importance of being able to see lower pole of kidney in skiagrams; operation—importance of avoiding nerves in making incision and in resecting twelfth rib—*anæsthesia dolorosa;* calculus detected high in ureter and removed; cut edges of ureter unite readily; postoperative comments.

HISTORY

The patient, a male aged twenty-eight years, was admitted to hospital September 24, 1914. The family history is negative. The past history revealed an attack of acute appendicitis four years ago—in 1910. After the appendix was removed the patient made an uneventful recovery and had no further trouble in the right iliac region until now.

The present trouble began eight months ago, in January, 1914, with a sudden and very severe pain in the right loin, which soon radiated downward and forward to the bladder: the pain was not felt in the testis. He had a fever of 99° F., but no chills, no nausea, and no vomiting. Four hours after the onset of the pain he voided very dark bloody urine. Thereafter he urinated frequently for about six hours. The pain subsided gradually. The next twenty-four hours there was soreness in the right lumbar region, and pressure and percussion over this area caused much pain. The stools have been normal throughout.

Two months later, in March, 1914, there was another similar attack, equally as severe as the first, and of about the same duration. The patient does not recall having had fever with it. These attacks recurred at intervals of about two months until four months ago, in May, 1914, when the intervals were shortened to about every three weeks. Lately the patient has felt weaker than usual between the attacks; but although the appetite has become poor, the weight has remained the same. The last attack began two days ago, on September 22, 1914, and lasted twelve hours. To relieve the pain he was given two hypodermics of morphin at intervals of about four hours.

As the attacks increased in frequency they increased in severity and in duration as well, and now persist for from twelve to thirty-six hours at a time. During the attacks and for some time afterward the patient's back always feels sore, and is tender to pressure over the painful area in the right lumbar region. The attacks have occurred in the early hours of the morning most often. Between the attacks he has noticed no disturbances of urination. The temperature has not been taken in every attack, but he thinks that it has often been high. During the paroxysms he has never noticed any pain or tenderness over McBurney's point, nor has there been tenderness in the right subcostal region. He has never been jaundiced, nor has he passed clay-colored stools. During the past four months the appetite has been poor, but he never felt distressed after meals. He has never passed a urinary calculus.

COMMENTS

Dr. Murphy (September 24, 1914): The intern's history is a fairly accurate statement of the case. The occurrence of an attack of acute appendicitis, for which the patient was operated upon preceding the onset of the initial attack of renal colic on the right side, is an interesting feature of the history, and arouses speculation as to whether this so-called appendiceal attack really was the cause of the disturbance or whether it was nothing more than the primary attack of renal colic masquerading as appendicitis. The intern's history throws no light upon this point, and it is an interesting matter for further investigation. Perhaps the surgeon did not find the acutely inflamed appendix which he was expecting. The late Dr. Maurice H. Richardson, of Boston, wrote upon this subject, as his last contribution to medical literature, under the caption: "The Error of Overlooking Ureteral Stone Under a Diagnosis of Appendicitis." This eminent surgeon cited numerous cases from his long experience in which this serious error had been made, chiefly because the possibility of calculus had not been taken into serious consideration. It is relatively easy to avoid this error if one only thinks of it, and is thorough in his diagnostic methods. On the other hand, acute appendicitis is occasionally wrongly diagnosed as renal colic, because of the presence of a few red cells in the urine. Where the acutely inflamed appendix lies directly over the ureter, the presence of an accompanying irritative ureteritis with a few red cells wandering into the urine is not such a very rare occurrence, and must be constantly borne in mind in making the diagnosis.

In order to call attention to the relative value of the symptoms of renal and ureteral calculi, I have had some statistics bearing upon this question placed on the board. They are tabulated from a large number of cases observed at the Mayo Clinic.

ANALYSIS OF SYMPTOMS OF RENAL LITHIASIS.—(*Braasch.*)

No. of Cases	Typical Pain in Kidney and Tenderness	Hematuria	Referred Pain
251	117	141 (56 per cent.)	30 to gall-bladder
			32 to lower abdomen
			56 to both sides of abdomen
			40 to affected sides of abdomen
			16 to non-affected sides of abdomen

Pain was absent or very slight in 21 cases.

In 26 pain was general throughout the abdomen.

In 131 cases of ureteral calculus pain was localized in the kidney in 107. Hematuria occurred with typical renal colic in 106 cases, with indefinite pain in 24, and without pain in 11.

Blood was found microscopically in the urine in 228 of the 251 cases.

In 500 consecutive cases without symptoms of renal trouble which were examined routinely 146 showed microscopic blood in the urine. Therefore, hematuria is not particularly indicative of renal pathology, unless associated with other symptoms.

Pus was found microscopically in 232 patients (93 per cent.). It has no special differential value, as it is connected with so many other lesions.

This table is a great aid in estimating the value of the individual symptoms. You may remember that, when speaking of the diagnosis of duodenal ulcer, I remarked that it was based upon not one symptom, but upon the interrelationship of a number of symptoms: to but a slightly less extent the same statement obtains for renal and ureteral calculi. In this series of 251 cases from the Mayo Clinic, pain in the kidney region, radiating down along the ureter, was present in only 117, or in less than 50 per cent. Hematuria was present in only 141 cases—just a little more than 50 per cent.—being absent, therefore, in almost half of the cases. Pain was referred to the gall-bladder region in 30 cases; to the lower abdomen in 32; to both sides of the abdomen in 56; to the affected side in 40,and to the non-affected side in 16, so you see that in a large percentage of the cases pain is present on both sides, either in the back or in the abdomen.

Another interesting observation is the frequency with which microscopic blood is found in the urine in the absence of any demonstrable lesion of the kidney. In 500 consecutive cases observed by Braasch microscopic blood was found in 146 patients who presented no renal symptoms.

My own experience is that the presence of red blood-corpuscles in the urine is not so unimportant a sign as might be inferred from the table. At the Mercy Hospital we are seldom able to demonstrate blood in the urine except when there is good reason to suspect the presence of some renal cause for it. A trace of albumin and a few red blood-cells may appear in the urine in one condition which has not been recognized sufficiently, and that is after repeated bimanual examination of the kidney. I first noted this occurrence in connection with the examination of patients with movable kidneys. Patients with this affliction usually have a trace of albumin and often a few red cells recorded on the urinary charts after they have been examined in clinic by myself and then by several students. I am speaking, of course, of patients who have shown no previous abnormality on urinary examination. On investigating this phenomenon more closely, I found that I was able to produce a trace of albumin in the urine with great regularity by squeezing and massaging the palpable kidney, and since that time I have often found this sign of great value in differentiating a tumor of the kidney from one lying outside of the kidney. When a kidney, the seat of a neoplasm, is repeatedly palpated by the bimanual method, a trace of albumin and some red cells appear in the urine almost invariably. Sometimes the ensuing hemorrhage is considerable and produces a distinct discoloration of the urine. This artificial hematuria is specially important diagnostically where the renal tumor had previously produced no demonstrable changes in the character of the urine, and in these cases it makes the diagnosis of renal tumor a practical certainty, because tumors in the vicinity can produce no such phenomenon unless adherent to the kidney, and unless the kidney and the adjacent tumor should be massaged at the same time, which is, of course, a theoretic possibility, but relatively easy to avoid in practice. Much has been written about "idiopathic" hematuria as an early sign of nephritis, and a very large percentage of these cases of hematuria of obscure etiology which have been followed for years have ultimately developed nephritis, just as have the cases of "lordotic" albuminuria. Passive congestion of the kidney from both local and general causes also produces blood in the urine. Renal and vesical varices, incipient tuberculosis, and several other local conditions have been demonstrated as the cause of "idiopathic" hematuria repeatedly. My experience, too, has been that blood in the urine indicates pathology in the genito-urinary tract. Surely any statistics on the subject of "idiopathic," "functional," or "physiologic" hematuria cannot be considered complete until the effects of palpation, massage, and of violent exercise can be excluded, functional tests of the renal parenchyma made, and the cases followed for a number of years in order to learn of their subsequent developments.

However, in renal calculus, just as in duodenal disease, it is the *combination of symptoms* that establishes the diagnosis. The presence of severe loin pain referred downward along the ureter, with accompanying hematuria, but without fever, composes the syndrome characteristic of nephrolithiasis. The skiagram verifies the diagnosis previously made by clinical methods. The sole finding which may claim to be pathognomonic is the shadow on the skiagram, but this shadow may be due to a phlebolith, an enterolith, a foreign body in the bowel, or an ancient calcified tuberculous focus in the kidney. There is one physical finding, however, which we have never found absent in a single case of *acute* obstruction of the ureter or its pelvis, and that is the reaction to fist-percussion (Murphy test) over the loin. When we suspect a renal cause for a colic we have the patient sit up, let him bend slightly forward, and place the left hand firmly over the healthy kidney: with the closed fist of the other hand strike the left hand firmly: this will bring no response from the patient. Then repeat the manœuver over the diseased kidney. If the renal capsule or ureteral pelvis be under tension, the patient springs up with a jerk, like a closing jack-knife, because of the severity of the pain evoked by this method. We have never seen this test positive in any condition that was not associated with increased tension upon the pelvis of the ureter, or the capsule of the kidney, or with a perirenal abscess. It makes a very positive objective demonstration of a purely subjective phenomenon—renal colic—and so corroborates the patient's statement regarding the pain. It is of value further because it is present in surgical conditions chiefly, and, therefore, when positive usually indicates operative intervention. Obstruction of the ureter at or below its pelvis is the most frequent cause of the pain evoked by fist-percussion, and when the obstruction disappears, and the tension on the renal capsule is relieved, the sign can be elicited no longer, although the pathologic cause of the obstruction, such as a calculus, may be still present. Fist-percussion is a sign of renal and ureteral calculus that is positive in the acute attack, and not in the intervals between attacks. Infarcts of the kidney, both sterile and septic, also present this sign during the

Fig. 232.—Preoperative skiagram showing calculus in the right ureter close to its upper end. Ureteral calculi are usually oval in outline, as here.

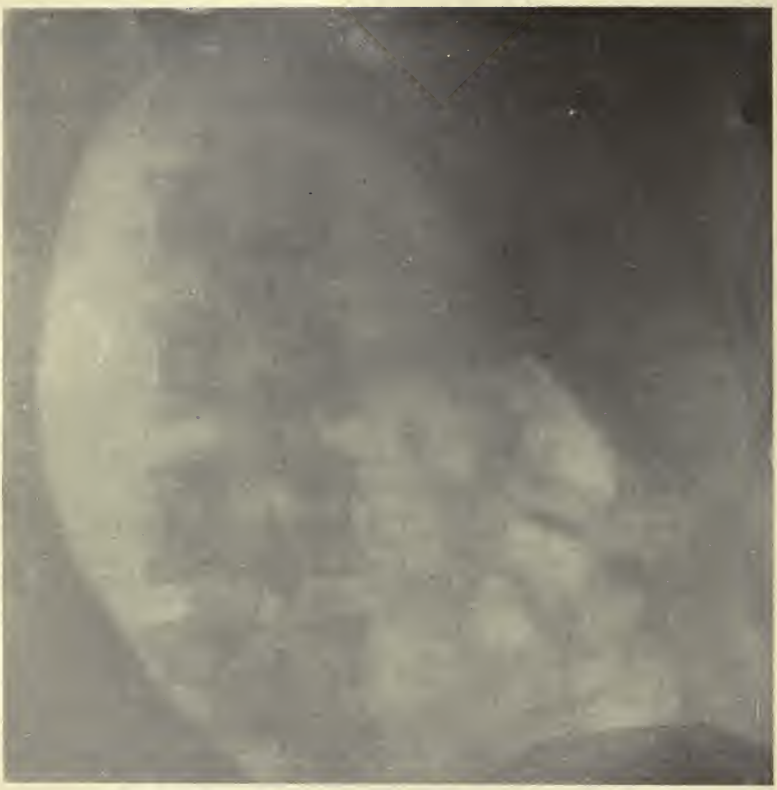


Fig. 233.—Preoperative skiagram showing calculus in right ureter.

acute stage, that is, within the first few days after their occurrence, because during that period the renal capsule is under tension. Let me call attention again to the fact that in only 117 cases of renal calculus was there typical renal colic with pain referred down along the ureter, and accompanied by local tenderness in the loin. In 21 cases renal calculus was present despite the fact that there had never been any appreciable pain or tenderness in the affected kidney: this statement means merely that in these 21 cases the calculi had never produced any obstruction to the urinary outflow. Pain signifies obstruction, retention and distention, while colic usually signifies that the obstructing body is in motion. When the calculus—for calculus is usually the cause of renal colic—stops moving, the colic stops, but the ache and local sensitiveness in the loin remain, unless the obstruction, too, be overcome, with relief of internal retention and the distention of the renal capsule.

The *x*-ray examination confirms the diagnosis previously made from the history and from the results of the physical examination (Figs. 232, 233). Recent reports from a hospital in London state that in 8 per cent. of the cases operated upon there the skiagram showed a calculus which was not found subsequently at operation; that is, there was failure to find the calculus in practically one out of every 12 cases. That is a high percentage of operative failures, if we interpret this observation correctly. According to this same hospital report the *x*-ray examination was negative for calculus in 6 per cent. of the cases in which a calculus was found at operation. Of course, these calculi, which the skiagram failed to demonstrate, were for the most part uric-acid calculi that have approximately the same specific gravity as the body tissues, and therefore throw a shadow no denser than the renal parenchyma itself.

In our work we always have two separate skiagrams made when we suspect the presence of a renal or an ureteral calculus, and we also empty thoroughly the bowels of these patients before the pictures are taken, thus avoiding error due to shadows cast by foreign bodies in the bowel. Phleboliths in the veins of the pelvis can be confused with ureteral calculi only when they lie very close to the ureter, and even then their true nature may be suspected from their shape and frequent multiplicity. A calculus when in the ureter is practically always oval in shape (Fig. 234), and, if large enough to obstruct the ureter, it is larger than the average phlebolith. The latter is also more circular in outline, as well as smaller than a calculus. The following conditions, seen in the skiagram, may also be mistaken for a calculus impacted in the ureter: small calcareous masses in the ends of the Fallopian tubes and ovaries; concretions in the appendix; ossification spots in the pelvic ligaments; concretions in bursæ of the pelvis; calcified epiploic appendage of sigmoid flexure, and a shrunken tuberculous kidney. In the relatively few cases in which the surgeon is really in doubt, he must catheterize the ureter with a metallic or a bismuth-impregnated catheter, which is impervious to the *x*-rays, and which, therefore, will show whether the suspected shadow lies outside of the ureter or inside of it—in the latter case obstructing it. Failure to find 8 per cent. of the calculi by operation when the *x*-ray indicated their presence seems to me too large a percentage of failures, and yet searching for a small calculus in the renal parenchyma is like searching for a fragment of needle in the palm of the hand, and those present who have had experience along these lines will recognize that an occasional failure is almost excusable.

In searching for a renal calculus it is important to orientate it in the kidney, and we can determine this point in advance if we have a well-taken skiagram to hand and if we know the varying morphologic appearances of calculi dependent upon their location in the kidney. The skiagram of the renal region is not properly made unless it show the outline of the lower pole of the kidney. The renal tissue may be differentiated from the extrarenal tissues because it is surrounded by a fatty capsule, the density of the renal parenchyma being greater than that of fat. Therefore, with the lower pole of the kidney in view as a landmark, it is possible almost accurately to estimate where the rest of the kidney lies, and so judge whether the calculus be located in the lower pole of the kidney, in its upper pole, in its middle, or in the pelvis of the ureter. Furthermore, calculi in the

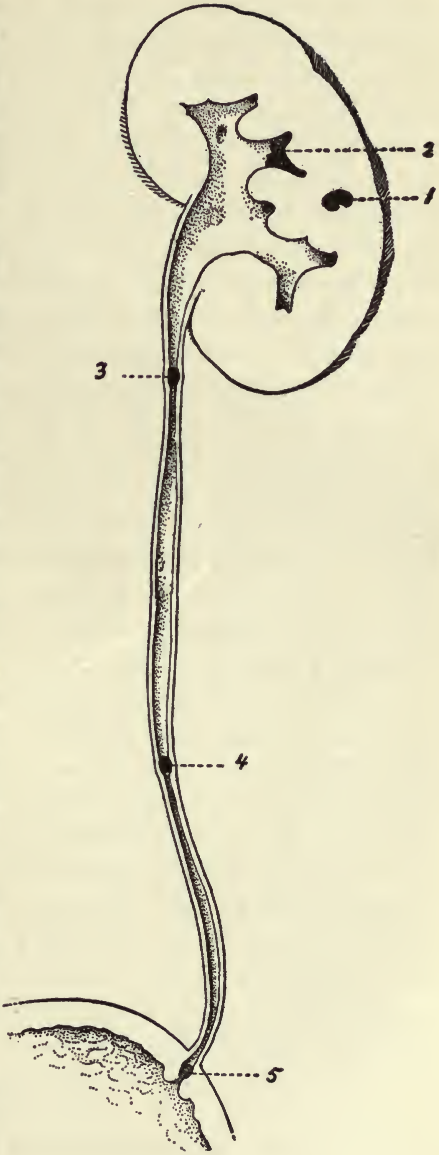


Fig. 234.—Diagram of upper urinary tract showing common situations of impacted urinary calculi and how their shape varies with their location: 1, Calculus in parenchyma of kidney. 2, Calculus in calyx. 3, 4, 5, Calculi impacted at points of constriction in normal ureter.

Calculi in the kidney are found at all ages, and with equal frequency in both sexes. No difference as to frequency between right and left kidney. In most cases limited to one kidney—bilateral in 11 out of 59 cases. Both kidneys not usually affected simultaneously. In 50 per cent. the kidney has a single stone; in the remaining 50 per cent. ordinarily 2 to 10 are found and up to 200. Proportion of renal to ureteral calculus is 2 : 3. When in parenchyma the calculus is usually small and round (1). Renal calculus is usually found in pelvis and when in a calyx is hour-glass shape (2). In ureteral portion of pelvis it is Y- or heart-shaped; and when in both pelvis and calyces, and filling them, it is coral-shaped, the stem being in the pelvis and the branches in the calyces.

Calculi in ureter are cylindric, because molded by the tube. When impacted they are most commonly (40.7 per cent.) arrested in the upper portion (3) and with about equal frequency in the middle (4) and vesical (5) portions.

Morris gives the position of ureteral impacted calculi found at operation in 44 cases. In 19 within two inches of kidney (3); in 15 just before or where ureter passes through vesical wall (5); in 11 somewhere about level of brim of true pelvis. Of these 11, in 3 the calculus was below the brim; in 2 *on* the brim; and in 5 just above the brim.

parenchyma are usually round, less frequently oval (Fig. 234). As was stated above, ureteral calculi are nearly always oval. Hook-shaped calculi are those lying at the bottom of the pelvis of the uteter, and dipping over into the neck of the ureter. Branching calculi are almost certainly located in the pelvis of the ureter, or in the calyces of the pelvis. Bearing these points in localization in mind, the search for renal calculi at operation is by no means the blind search that it was in the days before the use of the *x*-ray, or that it is liable to be if one operates without proper study of the case. Whenever you suspect the presence of a calculus in one kidney, do not forget to examine its fellow by both physical methods and by the *x*-ray. The local and systemic disorders which lead to the production of a calculus in one kidney are very liable to lead to calculus-formation in the opposite kidney as well. Only yesterday I examined a patient who had been operated upon sixteen years ago by the late Dr. Nicholas Senn. Dr. Senn removed a calculus from the right kidney. The skiagram now shows a calculus of similar size and shape in the left kidney. There has been no reformation of calculus in the right kidney.

According to these Mayo statistics, the pain was present in the non-affected side in 16 cases. If in these 16 cases one had examined the painful side alone with the *x*-rays, he would have overlooked entirely the calculus in the opposite kidney. The *x*-ray may also be the deciding factor in the operation—whether it is to be pyelotomy, ureterotomy, or nephrotomy. Surely, if one can avoid it, one will not incise the kidney for a calculus which can be reached by way of the pelvis. This latter plan I have followed uniformly since 1892, when I began it in my service in St. Joseph's Hospital. Furthermore, I always suture the ureter with fine catgut.

OPERATION

[Dr. Murphy made his usual lumbar incision.] I am starting this incision just below the twelfth rib. [Continues the dissection.] Here is the iliohypogastric nerve; above is the twelfth thoracic, and lower down the ilio-inguinal: I am carefully avoiding them in my dissection. To cut, or tear, one of these nerves produces a postoperative area of cutaneous anesthesia, which the patient is certain to complain of; and accidentally to include the nerve in a suture results in an area of either anesthesia or hyperesthesia, or in a combination of the two—*anæsthesia dolorosa.* [Dr. Murphy divides the twelfth rib to provide more room.]

On the right side the diaphragm is seldom below the level of the outer half of the twelfth rib and is, therefore, not much in danger of being incised with injury to the pleura when this rib is cut. [Dr. Murphy continues the dissection and exposes the kidney.]

Let the record show that the kidney is enlarged and situated high up beneath the diaphragm.

At last I am able to reach its upper pole and pass my finger around it. I have now enlarged the external opening enough successfully to deliver the kidney. [Dr. Murphy brings the kidney out upon the surface of the loin and retains it by the usual sling.]

We use these gauze strips as a sling to support the kidney in this position. The entire organ is much enlarged. I have between my fingers the pelvis of the ureter, but I can feel no calculus in it. Both the kidney and the pelvis of the ureter appear entirely free of calculi, just as we concluded from our study of the skiagrams. Now we shall examine the ureter, and I think we shall find the calculus at this point. Here it is, about an inch and a half below the pelvic end of the ureter; it is wedged down into the funnel which the upper end of the ureter makes, and that explains why it produced the obstruction. So tightly is it wedged, that it could not slip back. I am trying to make it slip back into the pelvis by milking the ureter, but it will not slip back, so that I must remove it as it lies in the ureter, and that will not be so very easy, because this portion of the ureter is not so accessible to us as the pelvis is.

I am exposing the ureter and bringing it up so that all in the room can see it. There it is, and here is the bulging which the calculus makes. I must palpate again to make certain that this is a calculus: it is a calculus and a very hard one. To prevent leakage I am compressing the ureter on each side of the calculus and am now making a longitudinal incision over the calculus. . . . It is still difficult to dislodge it, so firmly is it fixed in place. See how white it appears! It is a phosphatic calculus, apparently. Note these knobby little projections on it. Is it to be wondered at that they caused pain, and that the calculus was wedged in so firmly? We shall have no difficulty in uniting this ureter with an over-and-over stitch. The cut edges of the ureter heal more readily than those of any other tissue in the body, with the exception of the edges of the peritoneum. It was a little difficult to remove the calculus through this small opening, but I am glad that we were able to remove it without having to extend the incision farther downward, for we were able to lift the ureter up far enough to obtain fairly good access to it. I am now closing the ureter with a single over-and-over iodin catgut stitch.

Our next step is the closure of the wound, and we are drawing the muscles together with deep figure-of-8 stitches, in order to prevent the subsequent development of hernia.

Let the record show that on closing the wound the operator inspected the field to see that no sponges or instruments remained, and that Sister Victorine reported the sponges counted and correct.

POSTOPERATIVE COMMENTS

In a renal operation there is fully as much danger in leaving a sponge in the wound as there is in leaving one in the abdomen. Indeed, I think that the danger of a sponge producing subsequent complications is even greater in renal operations.

Sometimes a drop of urine may leak from the sutured ureter, but the last operation of ureterotomy which we performed was followed by primary healing without urinary leakage, and yet in that case we closed the ureter, as here, with but a single suture. As a precautionary measure, however, I am inserting this small rubber drain down to the site of the ureteral incision.

Let the record show that the kidney was much enlarged and lay high up in the loin, and that we had considerable difficulty in bringing it out through the subcostal incision, in spite of our having previously divided the twelfth rib to gain more room.

We used the curved subcostal incision instead of the downward prolongation of the usual lumbar incision, in order to secure easier access to this kidney, which was situated very high up.

Let the record show further that we exposed the last thoracic, the iliohypogastric, and the ilio-inguinal nerves early in the operation, but did not cut them in the original incision, and that we were careful also to avoid catching them in the figure-of-8 sutures with which we closed the wound.

[The usual copious gauze dressing was applied.]

[*N. B.*—The wound healed by complete primary union, and no urine escaped from it at any time. The wound was inspected on September 29th, and a few stitches were removed on October 2d. Four days later, on October 6, 1914, the drainage-tube and the remaining stitches were removed, and the patient was permitted to go home. He has remained well up to the present time.]

[A diagnostic talk on the symptoms and signs of renal and ureteral calculus was published in the CLINICS for October, 1914, pp. 871–873; and a mulberry and tunneled ureteral calculus, together with a skiagram, was reported in the CLINICS for April, 1913, pp. 287–293.]

RETROPERITONEAL SARCOMA — EXPLORATORY LAPAROTOMY

Summary: Clinical history of patient; examination; comments—diagnosis; *B. coli* infections form hard masses at times; operation—tumor exposed; postoperative comments—sites of lymphosarcoma and nature according to site involved—bloody transudate caused by sarcoma near serous tunics; value of Coley's serum in lymphosarcoma; use of arsenic in sarcoma, in certain types of connective-tissue tumors, and in sprue; origin and nature of sodium cacodylate; prognosis; natural history of sarcoma; fever necrotizes malignant cells; explanation of the paralysis in this patient; demonstration of patient two months later—tumor not in evidence.

HISTORY

The patient, a male aged twenty-one years, was admitted to hospital April 7, 1915. The family history is negative. The past history revealed an ischiorectal abscess one year previously, which was cured by an operation. The abscess had lasted three months. There is no history of injury.

The present trouble was first noticed nearly eight weeks ago, on February 15, 1915, and began acutely with extreme malaise and soreness in the left upper quadrant of the abdomen. There were no chills, nor was there fever, cough, or loss of appetite. He was constipated at the time. He was confined to bed for a week, at the end of which time he resumed work, but felt this constant sensation of malaise. After being at work three weeks, a numbness developed in the right great toe and he gradually lost control of the right foot. At present the numbness extends to within a few inches of the knee. About one week after the numbness in the toe developed he noticed a pain in the lumbar region of the spine, and this pain radiated around into the right hip and down the right lower extremity. By its persistence this pain keeps him awake nights.

Five days ago, on April 2d, the patient passed large amounts of urine, and the physician who examined it found pus and blood-cells. At present the urine approximates more nearly the normal, showing only four white cells to the 1/6 objective microscopic field. Since the onset of the malady, February 15th, the patient has lost from 15 to 18 pounds in weight. A leukocyte count made five days ago, on April 2d, showed 13,000 polymorphs. He has had no high fever, and the temperature upon admission at 3 o'clock to-day is 99.2° F. The leukocyte count to-day is 9000,—4000 less than the previous count,—and the hemoglobin estimation is 90 per cent.

Examination by Dr. Murphy on April 7th.—There are two masses—one on each side of the spinal column. The finger can pass between these two masses as one goes downward from the navel; ascending from the navel, they come very close together. They are hard—absolutely ligneous; they do not fluctuate, nor do they yield to compression like a psoas abscess would. This induration extends downward in the line of the rectus muscle, and seems to be fixed to the os pubis at the attachment of the rectus muscle. Rectal examination reveals that the pelvis is filled with a hard, indurated mass similar in hardness to those I felt above. The mass is anterior to the rectum: none of it is posterior. It does not involve the prostate. It is behind the bladder and between the bladder and the rectum, in the rectovesical pouch of peritoneum. The seminal vesicles are not involved. There is not the slightest fixation of the spine at any point. The skiagram does not show pathologic change in the body of any vertebra. The mass is a retroperitoneal condition that involves certain spinal nerves situated in its area, particularly the upper sacral trunks, and is probably a retroperitoneal sarcoma. The patient is advised to undergo an exploratory operation.

COMMENTS

Dr. Murphy (April 10, 1915): When you review the history and consider the pain in the back, the motor disturbances in the lower extremity, and the clinical detection of masses on both sides of the lower abdomen, the first thing you think of is Pott's disease of the spine; but when you ask the patient to bend his spine, there is perfect motion in the spine in all directions. If he had Pott's disease with granulation tissue compressing the spinal cord, he would not have a movable spine; that is proposition No. 1. Proposition No. 2 is to determine the nature of the hard masses in the abdomen. They do not fluctuate; they are smaller toward the sternum and diverge and enlarge as they go downward; as they pass upward they come over toward the mid-rectus muscle zone. The next condition to exclude is a mass of impacted feces. This tumor is as hard as wood; it is not movable; if it consisted of an accumulation of impacted feces it should be movable, but it is firmly fixed. Examination shows nothing abnormal in the chest, nor in the mediastinum, but rectal examination discloses that the whole field forms one firm, wood-like mass, as hard as bone,—ligneous, we called it,—and that it is situated anterior to the rectum, but not posterior. The mass fills all the available space in the pelvis, so that the examining finger is unable to get by it in any direction. The urinary bladder cannot be felt: it seems to be shut off from palpation by this hard, ligneous mass.

The next step in the diagnosis is the consideration of the urinary findings. Yesterday, April 9th, there was no sugar, nor albumin in the urine, and there were no casts, nor red blood-cells. Fifteen to 20 white cells were found in each microscopic field (1/6 objective), showing an increased number of leukocytes. Leukocytes in the urine are increased, first, by lesions within the bladder or elsewhere in the urinary tract; secondly, by lesions outside of the urinary tract, but juxtaposed to it. You find this leukocytic increase in women with pelvic cellulitis; you find it in men who have an infection in the pelvis, and in para- or perirenal abscesses not communicating with the ureter. An inflamed condition of the urinary tract and discharge from it would also give rise to this leukocytic increase. There is nothing additional to suggest an erosion in the urinary tract. A single specimen of urine to-day showed from 40 to 50 red blood-cells: at the office, forty-eight hours ago, there was no blood at all in the urine, so that the red blood-cells must have come after my bimanual examination.

These masses are extremely hard. Colon-bacillus infections and tuberculosis give rise to ligneous masses. Fecal concretions from the appendix may begin to bore their way to the surface and give all the appearances of a sarcoma; you cut through from one and one-half to two inches of infiltrated tissue and then get down upon the fecolith. I remember learning that lesson early in life, because I paid for it by an error in diagnosis and prognosis and the subsequent humiliation. Since learning it I have noticed that certain types of colon-bacillus infection give masses of infiltration resembling actinomycotic infections, but colon-bacillus and ray-fungus infections produce comparatively small masses, while here the masses of infiltration are very large.

After the physical examination and analysis of the history, the conclusion we reached was that we had to deal with a sarcoma, and we are about to perform an exploratory operation to be sure that the diagnosis is correct. If it is correct, then we have a definite plan of treatment to follow, but one which we are not justified in following to the superlative degree unless the diagnosis be proved beyond question.

Impacted feces could be just as hard as these masses, but they should be movable. I have never seen impacted feces that could not be displaced in some direction. A tuberculin test was made and was negative. These large masses to the left appear to be retroperitoneal sarcomata.

EXPLORATORY OPERATION

[Dr. Murphy makes the usual incision for a laparotomy.] There is the urinary bladder, pushed up by the tumor in the pelvis. You see why I could not palpate the bladder from below. There is the hard tumor—hard as wood—situated retroperitoneally and behind the mesentery. Now it is not necessary to excise a specimen of the tumor in order to establish the diagnosis: it diagnoses itself. In filling the pelvis the mass has pushed the bladder away out of the field. I would open that capsule of the sarcoma were it not for the fear of disseminating the disease throughout the peritoneum. The sarcoma is inoperable. Now we shall close the abdominal wall. We are making our usual ectropion of the peritoneal edges.

POSTOPERATIVE COMMENTS

What was the nature of the primary growth? I should say that it was a lymphosarcoma. Internally, lymphosarcoma begins only in a few distinct places where lymphoid tissue is present, as in the mediastinum, the retroperitoneal tissues, the subpleural tissues, and the tissue in the mesentery where the latter shades off into the retroperitoneal tissue. As the lymphosarcoma develops it may come up between the laminæ of the mesentery, pushing the bowel before it, and present beneath the abdominal wall hard, ligneous masses. You remember the importance we placed upon the ligneous hardness before opening the abdomen: this very hardness helps one establish the diagnosis. When the sarcoma begins in the mesentery of the small intestine, you will find involved practically the whole length of the mesentery, from the duodenojejunal flexure across the spine to the right sacro-iliac synchondrosis.

This tumor began on the side and remained on the side. There are no demonstrable metastases in the thoracic cavity. There is no fluid in the chest, and none in the peritoneal cavity. When the neoplasm increases in size, peritoneal fluid, which is so typical of malignant lesions involving the peritoneum or the subperitoneal tissue, will appear. Sarcoma causes a bloody transudate into the serous cavity of its neighboring field, and that bloody fluid is practically pathognomonic of malignancy. In tuberculosis of the lung or testis bloody fluid is found—though rarely—in the pleura and in the tunica vaginalis. Bloody fluid that is withdrawn during paracentesis of a serous cavity is always very suggestive, if not characteristic, of malignancy, and when withdrawn from the peritoneal cavity practically *always* indicates a para- or retroperitoneal sarcoma. Remember, I said *bloody fluid*, not blood.

Coley's Serum.—In a large percentage of the cases lymphosarcomata have shown more striking effects from Coley's serum than from any other form of treatment, although radium often causes rapid disappearance of this variety of sarcoma. This patient will be treated by Coley's serum together with diffuse exposures of high-tension x-rays over the abdomen. The Coley serum will be pushed as far as he can possibly stand it without his breaking down. Remember, this serum is a two-edged sword, and we are just beginning to learn some of the things for which we are indebted to Dr. Coley. I think that through Dr. Coley we are learning how to differentiate the cases which offer more striking results, and in these selected cases we are obtaining results that are worth while.

We have had two cases of malignant growths that we sent to Dr. Coley recently—one a carcinoma, the other a sarcoma. One of these neoplasms involved the bones of the face and pushed the eyes apart from each other. An operation was out of the question, and so we sent him to Dr. Coley. Three months later, upon receiving a photograph of the patient from Dr. Coley showing the effects of treatment, I could not recognize the man: the facial bones had receded into their normal positions. (See next case, p. 957.) That result was sufficient reward to the patient for having taken the treatment. Now in this case we shall start in to push the doses of Coley's serum to the limit of toleration.

Arsenic.—Associated with the Coley serum we give superlative doses of arsenic. Why? Because half a century or longer before we knew the specific relation between arsenic and the spirochetic infection, we knew that arsenic cured certain types of connective-tissue tumors. The old clinicians recognized this beneficial action of arsenic when they were giving Fowler's solution for tabes dorsalis—long before we knew anything about "606." Now we know that sodium cacodylate may be administered in colossal doses, hypodermically, of from two to ten grains every one, two, or three days. Billings was the first to use sodium cacodylate to promote absorption of the firm masses of connective tissue that were associated with pericarditis (*cor villosum*), and in this class of cases the drug had a striking effect. Long before it was used in that way we were using it for these neoplasms, but not in such large doses as we employ at the present time. When we found it had such striking effects upon connective-tissue neoplasms, we began using it in the treatment of the primary lesion of syphilis. I think that when given in large doses, daily and hypodermically, sodium cacodylate has a much more rapid curative effect than "606" has. If under its use a chancre does not disappear within ten or twelve days, you are not giving large enough doses. By beginning with two, three, or four grains daily and increasing the dose to ten grains every third day, you will be surprised how chancres just melt away within a week, and how the patients remain practically free from toxic manifestations of the drug.

While speaking of sodium cacodylate, it is interesting to record the experiences of Dr. J. O. Lunn, of Manila, P. I., who, on April 15, 1915, read a communication before the Manila Medical Society, entitled: "The Treatment and Cure of Sprue with Arsenical Compounds." Some of Dr. Lunn's conclusions were as follows:

"That either neosalvarsan or sodium cacodylate, when given intravenously or intramuscularly respectively, has the power of curing sprue.

"That apparently hopeless cases of sprue can be cured with sodium cacodylate.

"That sodium cacodylate may be given intramuscularly in very large doses without causing the slightest symptom of arsenical poisoning.

"That it is probably better to follow the suggestion of Dr. J. B. Murphy regarding the administration of sodium cacodylate in syphilis, *i. e.*, to give 4 or 5 grains of the drug at one dose, and not to repeat it within four days unless there are special indications therefor.

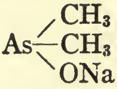
"That chemically pure sodium cacodylate only can be used.

"That administration of sodium cacodylate causes the patient none of the suffering, such as chills, fever, nausea, vomiting, and malaise, which some of the other arsenical compounds—namely, salvarsan and neosalvarsan—often cause."

[Sodium cacodylate has been referred to so frequently in these pages that a brief description of the drug will be given. The credit of introducing sodium cacodylate as a therapeutic agent belongs to Gautier, who, on June 6, 1899, reported a series of observations in conjunction with Burlureaux ("Cacodylates de Soude," *Bull. Gén. de Thérapeutique*, 1901, clxi, p. 524).]

Cacodylate of sodium is an organic salt of arsenic.

Formula:



$$\mathrm{As}\begin{matrix} / \mathrm{CH_3} \\ - \mathrm{CH_3} \\ \backslash \mathrm{ONa} \end{matrix}$$

It is a white, amorphous powder, soluble in water, tasteless and inodorous. The dose is from ⅓ to 1½ grains, although as much as 13 grains have been given daily for several weeks without producing toxic effects.

One and one-half grains of sodium cacodylate are equivalent in arsenic to about 1 grain of arsenous acid; 3 grains of sodium arsenate, and 100 minims of Fowler's solution.

Sodium cacodylate is best given *hypodermically* for ten days, followed by rest for ten days. Loss of appetite and elevation of temperature are indications for temporary cessation of treatment.

Gautier's original formula was:

℞.	Sodii cacodylat	gr. c (6.40 gm.)
	Aq. dest.	℥iij (90.0 c.c.)
	Acidi carbolici	♏j (0.06 c.c.)

Boil, filter through sterilized filter, and bring up to 100 c.c. (3 ounces) with distilled water. Each cubic centimeter contains 0.05 cgm. (⅚ grain) of the salt.

After injection, arsenic can be found at time of first urination, and its elimination continues for a month.

Gautier insists upon the necessity of administering the drug hypodermically.

PROGNOSIS

What is the prognosis in this case? It is hope: the hope that the patient will get better, the hope even that he will get well. I remember one of the early cases in which we used *x*-rays for another purpose upon a patient who also had sarcoma, and the sarcoma disappeared entirely. *X*-rays, sodium cacodylate, and Coley's serum seem to have a striking effect upon some types of sarcoma. Why do I say upon some types? Because we do not know much about the natural history of sarcoma except that there is a sarcoma for every type of connective tissue in the body. We know that some sarcomata grow rapidly while others grow slowly. We know that some of them are cured: some even by so slight a therapeutic agent as an elevation of temperature. Sarcomatous and carcinomatous cells do not withstand the degree of elevation of temperature that normal cells withstand, and are necrotized by high fever. Whether it is this elevation of temperature that so commonly follows the injection of Coley's serum that kills the malignant cells, or whether it is the effect of the mixed toxins that cures, we do not know, and to the patient it does not matter. We have at present in the hospital a patient who had an enormous inoperable sarcoma of the ilium. The tumor was so large that it measured from six to eight inches in diameter, but with the use of Coley's serum it has practically disappeared. The patient was a druggist—a very intelligent man—and he stood the reaction of Coley's serum very well. Were you to examine him at present you would scarcely find anything beyond some slight thickening of the ilium.

This patient's paralysis is due to the involvement of the sacral nerve-trunks in the neoplasm. His right lower extremity is paralyzed because the sarcoma has invaded the lumbosacral plexus, and this plexus is being subjected to compression because the reflexes have disappeared, showing that it is a peripheral lesion, and not a cord lesion higher up than the sacral centers. In the history the patient said that he could not walk because the right leg was paralyzed. He has no reflexes present to-day—an indication that the neoplasm involves the nerves between the presiding cell-bodies in the spinal cord and the periphery. If the reflexes are present and exaggerated, the lesion is situated proximal to the presiding cells in the cord.

RECAPITULATION

Let the record show that in the right side of the abdomen, extending up to the kidney and down into the pelvis, there was a firm, ligneous mass with a vascular surface, immovable and fixed to the bones of the pelvis; that on the opposite side there was the same condition extending up along the side of the spine, leaving a sulcus in the middle that resembled in appearance, but not in size, a cerebral sulcus.

When you look at the tumor *in situ* it reproduces the shape of the cerebellum. Extending deeply into the pelvis along the side of the pubic bone was this firm, round thickening; it did not, however, begin in the pubic bone. It was this fixed infiltration of the neoplasm, as it went down into the right side of the pelvis and involved the sciatic nerve, that caused the paralysis of the lower extremity.

Let the record show further that it was deemed inadvisable to remove a portion of the tumor for diagnostic purposes, lest the division of its capsule would expose the peritoneum to the semination of malignant cells which, if implanted, could spread rapidly over the raw peritoneal surface; that the bladder was pushed entirely out of the pelvis upward to above the symphysis pubis, so that it came into the field when I was opening the abdomen, necessitating my entering higher in order to avoid injuring the bladder.

Let the record show that, on closing, the operator examined the peritoneal cavity to see that no sponges or instruments remained; and that Sister Victorine reported the sponges counted and correct.

Dr. Murphy (June 8, 1915, two months after operation): Here is a striking case of sarcoma of the lymphoid type. You see that the tumor has practically disappeared. As to the permanency of cure, we are uncertain, but the result is striking in a case in which operative removal of the neoplasm was out of the question.

[Cases of retroperitoneal sarcoma have been published in the Clinics for August, 1912, pp. 537–544; December, 1914, pp. 1215–1223. With the latter case is included a sketch of the tumor *in situ* on p. 1221.]

INOPERABLE RECURRENT CARCINOMA OF NASOPHARYNX INVOLVING BOTH SUPERIOR MAXILLÆ, ETHMOID, FRONTAL AND MALAR BONES — INJECTION OF MIXED TOXINS—DISAPPEARANCE OF NEOPLASM UNDER FIVE WEEKS OF TREATMENT

By William B. Coley, M.D.
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The patient, a male aged thirty-eight years, was admitted to the General Memorial Hospital in New York city on January 27, 1915. The family history is negative. The patient has a congenital cleft-palate.

Five months previously, in August, 1914, he first noticed difficulty in breathing through the nostrils. On August 15th he consulted Dr. J. H. Thorpe, of Owensboro, Kentucky, who found a tumor in the nasopharynx, behind the soft palate. On August 18th two specimens were removed from the neoplasm, both of which, however, were lost during transit in the mails. Two weeks later another specimen was removed, and microscopic examination showed it to be nothing but simple adenoid tissue. Clinically, Dr. Thorpe regarded the trouble as malignant. On September 3d he operated upon the patient, removing as much of the tumor as he could reach. Recurrence took place almost immediately, and the disease extended upward into the region of the bridge of the nose and frontal sinuses. A second operation was performed by Dr. Thorpe on December 12, 1914, with electric wire, cautery, and curet. The operation was an extensive one, though incomplete, and the patient lost much blood. The tumor grew more rapidly after the operation than before. The patient's health failed rapidly and the pain became more severe, requiring ¼-grain doses of morphin for relief.

In October, 1914, the patient noticed that his jaws did not come together, and that the upper jaw in the region of the cleft in the hard palate had begun to spread; he was no longer able to wear a plate. On December 28th he consulted a physician of Louisville, Kentucky, who stated that the tumor was undoubtedly malignant, *probably carcinomatous*, but that it was too extensive for operative treatment, and advised the patient's wife to take him home and make him as comfortable as possible, thereby promoting euthanasia. He continued to grow worse, and by the middle of January was confined to bed. He began to have hemorrhages from the tumor, sometimes losing as much as several ounces of blood at a time.

On January 25, 1915, he was taken to Chicago to consult Dr. J. B. Murphy. The latter had an *x*-ray photograph taken of the patient's head, but considered the condition entirely inoperable, and referred him to me for toxin treatment.

Upon admission to my service physical examination showed that the patient was very weak and anemic, and unable to walk without being supported. He had severe pain in the head, and the pain was specially marked in the frontal region, and required ¼-grain doses of morphin for relief. The whole contour of the forehead and upper portion of the face and nose was markedly distorted (Figs. 235, 236). There was a tumor involving both nasal cavities, both superior maxillæ, and the ethmoid and sphenoid bones, producing a great broadening of the nose, particularly marked at the bridge of the nose (which was two inches in width), and bulging of the whole frontal region. The right malar bone was much more prominent than the left, but both were enlarged. There was also marked exophthalmos of the right eye, with dislocation of the ball outward for one inch or more, causing inability to focus; he had not been able to read for more than a month. The tumor extended down as far as the upper lip. The patient had a complete congenital cleft-palate—*an embryonal defect which, based on the Cohnheim theory, might have had an etiologic influence in the development of this malignancy.*

The superior maxillæ had been separated by the tumor, creating a space one inch wide in the center, where one of the incisor teeth had been drawn. Through the cleft in the soft palate a large, fungating tumor could be seen, occupying the whole space between the soft palate and the pharynx. This tumor bled very easily, the patient losing more than one-half pint of blood on some days.

In view of the very advanced stage of the disease and the poor general condition of the patient, I did not believe that the toxins offered any reasonable hope of success, and advised his wife to take him to Kentucky, without treatment, believing that he could live but a very short time. She insisted, however, upon a trial of the toxins, in spite of the hopeless prognosis which I gave. Hence, on January 27, 1915, treatment by the toxins was begun, the initial dose being ½ minim, which was injected into the pectoral region. The dose was increased by ½ minim each day.

On January 29th there was slight bleeding from the tumor behind the palate. The hemorrhages increased, and on February 3d the patient lost 5 ounces of blood in the morning and 2 ounces in the evening. On February 5th there was considerable bleeding, which was only partly controlled by spraying with adrenalin and by packing. On February 6th an injection of 10 c.c. of horse-serum was given subcutaneously, after which the hemorrhages temporarily became much less until, on February 11th, when he lost 6 ounces of blood in the morning and 4 ounces later in the day. Another dose of 10 c.c. of horse-serum was administered. On February 12th he lost 12 ounces of blood, and on February 13th he lost 5 ounces at one time and 2 ounces at another. The bleeding continued at intervals of from twenty to thirty minutes during the day.

On February 14th there was slight bleeding from the mouth. By this time the dose of toxins had reached 6 minims, causing a slight fever of 99° F., but no chill. After the first week the dose was increased by 1 minim a day, and on February 16th, with a dose of 7 minims, the first chill occurred, which lasted for twenty minutes, and was followed by a fever of 101° F. From this time on he had no further hemorrhages.

The pronounced local and general improvement, which had been noticeable at the end of one week's treatment, continued. There was marked diminution in the size of the external tumors, as well as decrease in the size of the tumor in the nasopharynx. Eight minims given on February 19th caused no chill, nor did the next three doses of 9½ minims each, given on the twentieth, twenty-first, and twenty-third of February cause a chill. On the twenty-fourth, 9½ minims produced a chill. On the twenty-sixth, 10 minims—no chill; on the twenty-eighth, 10½ minims—no chill. On March 1st the same dose as on the preceding day, injected into the pectoral region, was followed by a very severe reaction; the patient had not entirely recovered from the depression of the preceding day. One hour after the injection he had a chill lasting forty minutes; one hour after this a second chill occurred which lasted an hour; the temperature rose to 104° immediately after the first chill; one hour after the second chill it was 104.6° F. The patient was in a state of collapse; instead of being cyanotic, he became unusually red. The pulse went up to 136 and was very weak; respiration, 32. He was not given any stimulants. Two hours after the second chill the fever fell to 102° F., and the next morning both pulse and temperature had fallen to normal. The patient was very weak, but was up and about the room.

On March 3d, two days later, a very remarkable change occurred in the tumor, as well as in the contour of the face *Cf.* Figs. 237 and 238. The eyes were much closer together—nearly the normal distance apart; the exophthalmos, which had become much diminished, had practically disappeared; the bulging in the frontal region had entirely subsided, and for the first time in three months the jaws came together in their normal position. The patient was then given a respite from treatment for two or three days, after which the injections were resumed in smaller doses—5 minims. He received 22 treatments in all while in hospital. The hemoglobin, which was 80 per cent. on admission, fell to 68 per cent. by the time of discharge. The improvement

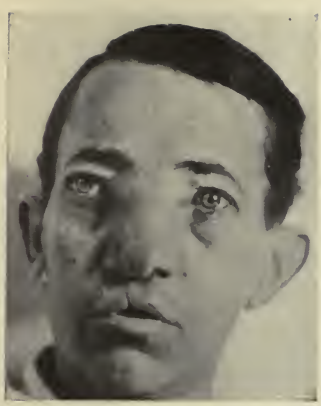

Fig. 235.—February 12, 1915.

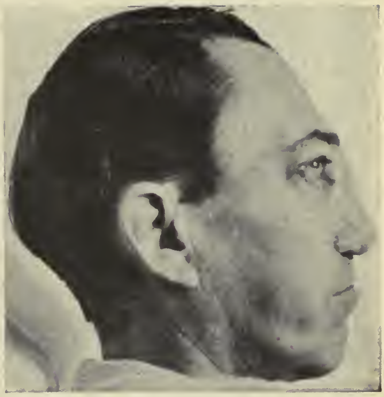

Fig. 236.—February 12, 1915 (side view).

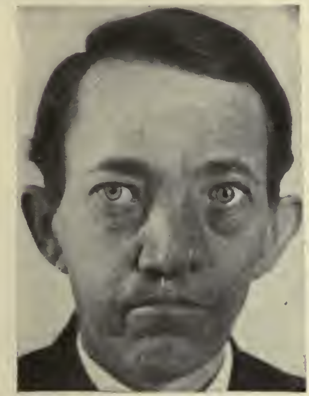

Fig. 237.—March 18, 1915.

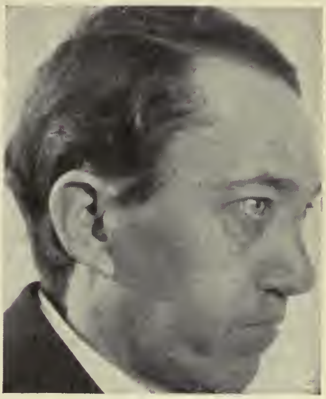

Fig. 238.—March 18, 1915.

Cancer of Nasopharynx Disappearing Under Treatment with the Mixed Toxins (Coley, in *Annals of Surgery*).

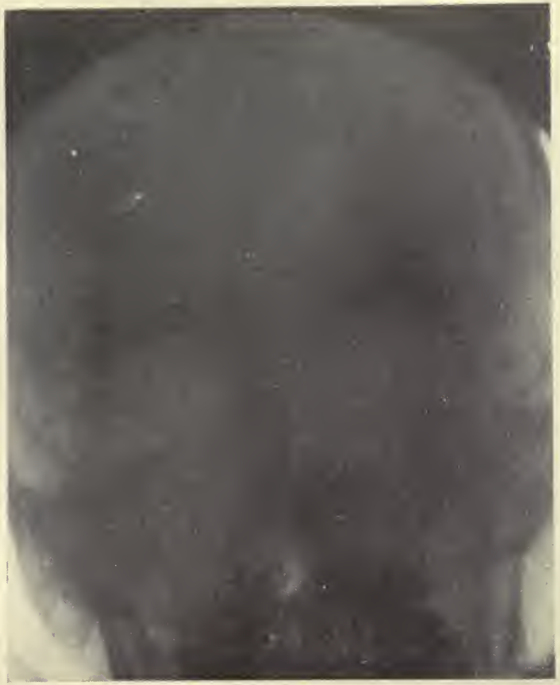

Fig. 239.—February 12, 1915.

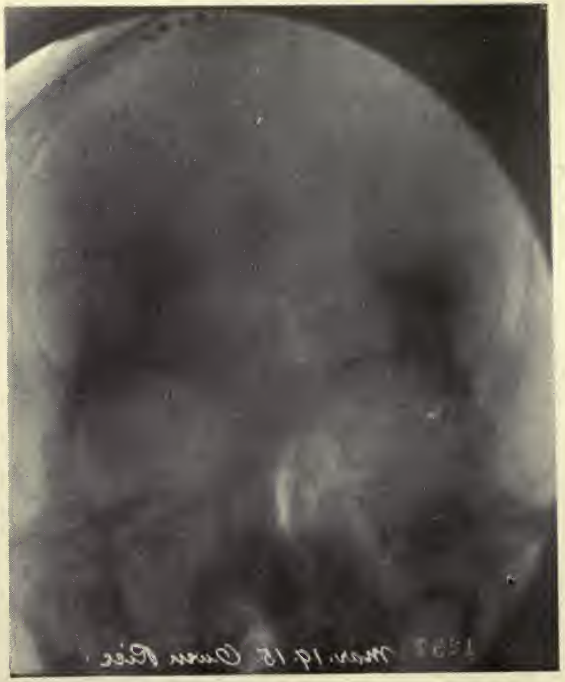

Fig. 240.—March 20, 1915.

(Coley, in *Annals of Surgery.*)

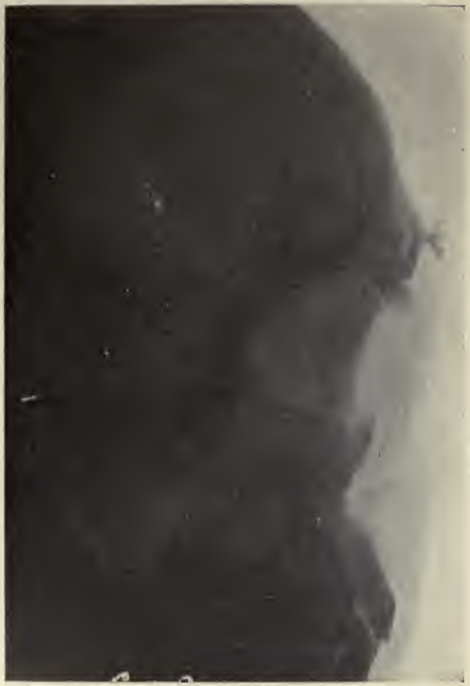

Fig. 241.—February 12, 1915.

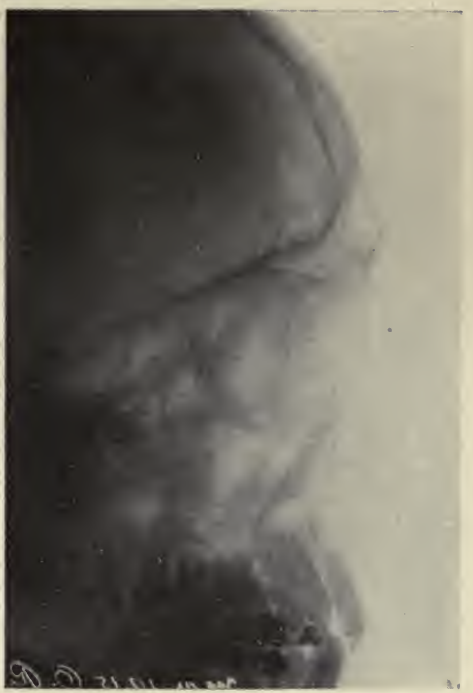

Fig. 242.—March 20, 1915.

(Coley, in *Annals of Surgery.*)

continued; he gained in weight at the rate of nearly a pound a day. On the day of discharge from hospital, March 9th, the following note was made by the resident surgeon, Dr. G. R. Dempsey:

> "Patient discharged March 9th. The swelling of the bridge of the nose between the eyes has entirely disappeared; the eyes appear to be in a normal position; the cleft in the palate is much narrower; there is now only room for one tooth in the gap of the upper jaw (on entering the hospital this gap was one inch wide); through the opening in the palate only smooth, clean tissue can be seen; there is no evidence of any tumor; the patient's teeth now meet properly, for the first time in months. Except a little thickening over the nose, the signs and subjective symptoms have disappeared entirely. The patient has gained markedly in weight and in strength."

For the last two weeks the patient has been able to breathe through one nostril, and for the last week through both, for the first time since August 15, 1914.

Three *x*-ray pictures were taken: the first one, taken before the toxin treatment was begun, showed only a general confused blurring of the whole plate, the outlines of the different bones being completely lost.

The second skiagram was taken on February 12th, and showed extensive destruction of the superior maxillæ, the turbinates, the ethmoids, the vomer, etc., extending to the sphenoidal sinus (Figs. 239, 241).

The third and last skiagram, taken on March 20th by Dr. Holding, showed remarkable changes, the different bones having nearly recovered their normal outlines. The patient has had toxins in small doses of from 3 to 4 minims daily since he left hospital, and has gained 14 pounds in weight. The contour of the face and nose is entirely normal, and no trace of tumor can be found (Figs. 240, 242).

While it is unfortunate that we have no microscopic report of the tumor, the history of rapid growth and the clinical features, together with the *x*-ray photographs, admit of no reasonable doubt that the tumor was a very malignant one. The negative Wassermann reaction strengthens this view still further.

LATER NOTES

April 21, 1915.—A slide of the original specimen, removed for examination in August, 1914, was obtained from Dr. Hays, of Louisville, and examined by Dr. James Ewing, whose conclusions are as follows:

"The exact nature of the tumor it is impossible to state, but *the general appearances are those of a carcinoma* with poorly differentiated epithelial cells, and origin from the lining cells of the mucosa seems probable. *Of the malignant characters of the process there can be no doubt.*"

April 26, 1915.—A recurrence the size of an English walnut was found behind the angle of the right side of the mandible, beneath the anterior border of the sternomastoid muscle. The patient was put on the mixed toxins, and had no chill until 25 minims were reached.

July 1, 1915.—The patient's appetite has returned. The separation in the jaw is narrowing (it is only 3/8 inch now), and the enlarged lymph-node is one-fourth the size it was on April 26th. The patient is receiving 5 injections a week, and is now taking 27 minims of the toxins at one dose.

During his second stay in the hospital he had three *x*-ray treatments.

August 3, 1915.—Examination to-day shows no trace of the tumor. The exophthalmos has disappeared entirely: the enlarged lymph-nodes on both sides of the neck have also disappeared. The separation of the superior maxillæ has reduced to normal, and the patient breathes freely through the nostrils. He has regained most of the lost weight, and feels as well as usual. During July toxins were given three or four times a week, the largest dose being 32 minims. He was discharged from hospital July 31, 1915.

Dr. Coley: This case is specially noteworthy and instructive for the following reasons:

1. After a very far-advanced and inoperable malignant

tumor had apparently disappeared under toxin treatment, a quick recurrence followed a reduction in the size of the dose from 10 minims to 2 or 3 minims.

2. The recurrent tumors, both primary and metastatic, grew rapidly, and showed no signs of control until the dose of toxins had been increased to more than double the amount tolerated during the first period of treatment.

3. Under these large doses and severe reactions all evidence of both primary and metastatic tumors again disappeared, and the patient's general health was restored.

This case seems to conform to my opinion, previously expressed, that success or failure in a given case depends largely upon—(1) the proper, but often very difficult, adaptation of the dose of toxins to such cases, and (2) continuation of the treatment over a long period.

METASTATIC ARTHRITIS OF KNEE-JOINT —VICIOUS FLEXION-CONTRACTURE—TENOTOMY OF BICEPS FEMORIS WITH CORRECTION OF DEFORMITY. TALK ON AUTOSENSITIZED AUTOGENOUS VACCINES

Summary: Clinical history of patient; comments—arthritis—its relation to ankylosis; subluxation of knee-joint and great contraction of biceps; importance of deep ether anesthesia; danger to vessels in straightening a long-flexed leg; operation—tenotomy of biceps; recapitulation; postoperative comments—the metastatic arthritides; use of autosensitized vaccines.

HISTORY

The patient, a female aged forty-one years, was admitted to hospital December 9, 1914. The family history was negative. She had typhoid fever at the age of twelve years, but has had no sequelæ. Postoperative menopause occurred in 1907, following a "colostomy."

The present infection started eight months ago, in the spring of the present year, from a callus beneath the great toe of the right foot. From five to eight days after paring the callus, making it bleed, the patient noticed a small but painful swelling on the heel, followed by abscess-formation. The abscess was opened and discharged until it healed in the latter part of July.

On August 13, 1914, shortly after this abscess was cured, the patient went down town at noontime and soon was seized with severe pains in all joints, both large and small. The calf of the left leg and the outer side of the right knee were tender. She arrived home at 4 P. M. with a chilly sensation, a severe headache, and a fever of 103° F. Then there followed severe pains in the right knee, to obtain relief from which she flexed the leg upon the thigh, and in this position it has continued remaining. She has been in bed since the onset of the trouble.

Four days later, on August 17th, the pains in other joints than the right knee became more intense. The pain was *general* throughout all the joints with the exception of the right knee, the right wrist, the left ankle, and the left thumb, in which joints the pain was more *localized*. The pain in the right knee has continued, but that in the other joints subsided. At present the pain in the right knee is aggravated by moving the leg, and the knee is tender to touch and swollen.

COMMENTS

Dr. Murphy (December 12, 1914): What is there interesting in this history? Just take the thread of the story, unravel it, and weave the course of the infections. It began first with an infection of the toe, and that infection extended through the lymphatics of the foot and formed an abscess in the heel. When this abscess was practically cured, the patient developed a *chill*, headache, high fever (103° F.), and localization of the pain around the right knee-joint and in the calf of the left leg. That was on Saturday. The next Tuesday all joints became involved, completing the typical story of a metastatic arthritis, and including the typical occurrence of an episode of even more significance than the metastatic arthritis, namely, the inception of the attack *with a chill. A metastatic arthritis or "rheumatism" that begins with a chill, as a rule and as a law, to which there are, however, exceptions, results in ankylosis of one or more joints.* This proposition comes up over and over again. If the infection is of low type and metastatic arthritis develops without any chill, the sequence of ankylosis commonly is absent. Even though pyarthrosis develop, if the pus be evacuated immediately by aspiration or by a small incision, and *if no drainage-tubes be inserted, nor the joint irrigated*, the joint might heal without ankylosis. But this patient's joint infection began with a chill, after which this distressing state of affairs developed. Even with the extension on, the pain was so intolerable that she could not straighten out the leg. The leg has been flexed upon the thigh but a short time, and upon this fact we base much hope of obtaining good reduction without open operation in the joint.

Last Wednesday we operated upon a young man who contracted the infection twenty-three years before coming to us, and had the resulting flexion. Nothing could be done for him except the radical open operation into the knee-joint.

Note the subluxation and enormous contraction here now. This is the biceps tendon, and it forms the external hamstring. There is never so much tension on the internal hamstrings as on the external hamstring. These internal hamstrings are attached to the tibia and the external hamstring is attached to the fibula.

You cannot do anything with these cases without the most profound anesthesia: that is why we do not give them gas.

To-day we shall endeavor to bring the leg down straight, with or without division of the internal hamstrings high up. If we can accomplish that manœuver, then we shall apply a plaster-of-Paris dressing to retain the leg in extension, hoping to secure ankylosis in the straight position. If the leg should remain in this position of flexion, bony ankylosis in flexion will result; she will then never be able to use it, and it might as well be amputated. That is a very serious and very grave situation. The first day after operation there will be a great deal of pain.

In straightening a leg that has been flexed for a long time, there is always the danger of injuring the popliteal vessels. In this case injury to the vessels should be slight, because only a few months have elapsed since the flexion appeared. It was entirely different in the case we operated upon Wednesday, because that patient had had the condition twenty-three years, and the cicatricial tissue in the biceps was so great that it had produced not only flexion of the leg to an acute angle, but also rotation of the leg until the foot was at a right angle with the long axis of the limb. However, through an open incision, after elongating the biceps tendon by seven inches, and then dividing the external lateral ligaments and freeing all other attachments of the knee-joint, we succeeded in bringing the limb out straight. (See next case.)

OPERATION

[Incision and exposure of field.] Now we shall divide the external hamstring tendon. In this operation of tenotomy it is important to make the division high up above the fibula, inserting the knife just at the inner border of the muscle, because of the danger to which the external popliteal nerve is exposed in this situation. . . . You can see the location of the nerve as it slips beneath my finger. This peroneal nerve can be paralyzed in two ways: first, by cutting, which is infrequent; and, second, by stretching, which occurred in the case operated upon Wednesday. I am performing the tenotomy exactly as in the subcutaneous method, so that some of the biceps muscle will remain intact and the rest will spread. I am compressing the muscle with thumb-pressure so as to tear through it, instead of cutting it. You can now see that the tendon has been cut and you see the ligaments stretch. We have succeeded in bringing the leg around in good shape. We must not let the patient awake until the entire plaster-of-Paris dressing has been applied. This dressing must include the thigh and hip. Note the enormous tension that is put upon the external popliteal nerve. If we can get ankylosis of this knee-joint in extension, we shall be just about as happy as we could be. Restoration of motion by arthroplasty will then be available to the patient in the near future, if we succeed.

RECAPITULATION

Let the record show that before dividing the external hamstring it was impossible to extend the knee; that we performed subcutaneous division of the biceps muscle above its tendinous formation; that we then tore through the remaining portion of the muscle with thumb-pressure, just as when we tear the adductors in performing an operation for spreading and abduction of the lower extremity.

POSTOPERATIVE COMMENTS

Now let us review the metastatic arthritides as they come to us. When one sees one case of this kind, or one hundred cases of that kind of metastatic arthritis, he wonders that the connection between the primary infection and the secondary metastases had not been observed by the profession and established centuries ago. Still the fact remains that millions of necropsies had been made before the relation of the appendix to disease in the right iliac fossa was established. Every time the abdomen was dissected, and every time necropsy was performed for septic peritonitis, the handwriting on the wall indicated that the peritonitis originated in the appendix, and still we went on, century after century, before the facts were established. A similar comment might justly be made when one considers the length of time we were all performing laparotomies in the right hypochondrium for gall-stones and gastric ulcer before Mayo recognized the frequency and significance of the duodenal ulcer. Still, in duodenal ulcer one does not have the gross external manifestations that occur with appendicitis or with these arthritides. One of the reasons why these lesions were not associated with cause and effect earlier, and one of the points I endeavor to emphasize in cases of this class, is that we lose the connection because of the necessary time that elapses between the primary infection and the secondary arthritic metastases. In but one type of metastatic arthritis, so far as we have been able to determine, is there early metastasis during the actively progressing infection of the primary lesion—it is only in the streptococcic infections that early joint-metastases appear, *i. e.*, within the first twenty-four or forty-eight hours from their onset. Why? Because streptococci invade the lymph-vessels and filter through the lymph-nodes directly into the blood-stream, and metastasize early into the joints. Staphylococci metastasize at definite periods after their primary local invasion; pneumococci and the diplococcus of Neisser metastasize at their own definite periods. All these metastasize when the infection is far advanced in lysis, or in a recrudescence of the infection. While the Neisserian disease is in the height of its activity locally it does not metastasize: the secondary joint manifestations occur as the disease subsides, while the urethritis is either in the process of repair, or near the completion of repair. To express it briefly, when these metastatic manifestations occur, the disease has passed the acme of the acute severity of the primary infection.

That is why a man who had influenza ten or twelve days previously, while up and about doing this, that, or the other thing, gets a chill one day and then "rheumatism." It is not because he was up and about, or exposed; it is because it was *the cycle-time* for the metastases to take place, and the fact that he was doing various things, and had a chill, was merely a coincidence.

The single metastatic arthritis that we *did* recognize for a long time, and the one over which a very warm controversy was waged, was the Neisserian "rheumatism," or the Neisserian joint. We observed the fact that the patient practically never developed the Neisserian joint before the eighteenth day, and then from the eighteenth to the twenty-second day in more than 95 per cent. of the cases in which a joint was involved. Upon reviewing the histories of these cases, when joint manifestations appeared between the eighteenth and twenty-second days, you will find that in one case the man jumped over a fence; in another he sat in a draft, etc. Such incidents were merely coincidental: it is because that was about the regular time for metastases to occur.

The same holds true of the typhoid infections in relation to the time of metastases. We practically never see a case of typhoid osteitis, of typhoid osteoperiostitis, or of typhoid arthritis before the end of the third week of the disease. These metastatic lesions occur between the third and fifth weeks, when the typhoid ulcer is undergoing repair.

The older men present—those who lived in the pre-antitoxin period—will recall that in treating a case of diphtheria they were happy when the membrane came off, as then they considered the case advanced toward recovery. They said: "I shall come back tomorrow and that will probably be my last visit." That night they were called. The patient was reported as having difficulty of deglutition, or else dyspnea. They went back to see the child which they had left in the morning greatly improved, and with only a raw surface upon the throat in place of the membrane, to find in the evening that the pulse was 140, 160, or 180, with the child perfectly lucid, but with metastatic cardiac mycoses; the patient was usually dead before morning. Or if deglutition were involved, the glossopharyngeal nucleus was the center of a metastatic infection. I had that shocking experience more than once. Again, in the other type of diphtheria, the parents sent for the physician on the second day after the membrane had fallen off and said to him: "When he swallows milk it comes out through the nose." This signified a paralysis of the glossopharyngeal nerve—a metastatic intoxication of the nerve which bore the same relation to the falling off of the membrane that the cardiac mycoses bore.

What I want to emphasize is that there is a definite time relationship between the primary infection and the appearance of the metastases. What I want to dilate upon still further is the chill that ushers in the joint manifestations as a secondary indication that the infection has passed into the blood; and that if after the chill the infection should focalize in the joint, in all probability the ultimate result will be ankylosis.

AUTOSENSITIZED AUTOGENOUS VACCINES

It is your duty carefully to watch the primary lesion, and to prepare an autogenous vaccine the moment the manifestations of joint-infection appear. The earlier it is prepared and administered the more effective the vaccine. You can easily make a culture from the patient's blood in the early stage, but not so easily in the later stage, for the culture must be made while the microörganisms are still in the blood-stream. We have gone still further with our vaccines. We believe the reason we have had so little effect from vaccines in some cases—aside from the colon bacillus and the pyocyaneus groups of infection—is that the culture-medium has been faulty.

I refer frequently to the experiments of Rosenau, because they are the most elucidating experiments that have been carried out in modern bacteriology. Rosenau found that by cultivating a single strain of *Streptococcus viridans* in six different media, by injection into animals he could obtain as many different types of streptococcic disease, and each type from each medium uniformly. From one strain he could produce typical ulcerative endocarditis; from another, gastric or duodenal ulcer; from another, toxic myositis (muscular rheumatism); from another, typical pneumonia in which the organisms resembled the diplococcus of pneumonia, and so on through the six media. It was these experiments, applied to our knowledge of autotransplants of tissues, that led us to the conclusion that *in order to obtain the best results from vaccines, not only should the microörganisms come from the patient himself, but this autogenous strain should be cultivated upon the patient's own blood-serum, thus giving an automedium.* This conception resulted in our production of the autosensitized vaccine, which we have been employing for some months.

We reached that conclusion from the fact that in the course of our surgical work we found the autotransplant to be the only effective transplant—the only transplant with regenerative, stimulative, and osteogenetic influences. We know that cold-storage bone, from a foreign source, when inserted into the medulla, acts as a support, but has no osteogenetic influence, and eventually will be wholly absorbed. The osteogenetic force comes from the patient's own bone, and no more from a heterotransplant than from a magnesium or a metal tube placed in the medulla.

From Rosenau's experiments, as well as our own observations with bone-transplants, we concluded that the best results, therefore, with vaccines should be obtained by using the same microörganisms that produced the infection, and cultivating them upon the patient's own blood-serum. *We believe the autosensitized vaccine is a decided advance in the vaccine treatment of disease.* We are treating practically all our acute cases in that way.

If we cannot isolate from the patient the microörganisms that are causing the trouble, guided by the history of the disease we culture upon the patient's serum foreign microörganisms that, as a class, were etiologic in producing the disease in question. For instance, if the patient's arthritis follows typhoid infection, we cultivate *B. typhosus*, obtained from other sources, upon foreign media *first*, and *then* upon his own blood-serum, thus approximating as nearly as is possible autosensitized material for injection. Upon the whole, we are very much impressed with the vaccine treatment—especially the autosensitized—

from an empirical standpoint. The dose of either autovaccines autosensitized, or homovaccines autosensitized, that can be administered and tolerated, is enormously greater than that of heterocultivated vaccines.

Two, three, or even ten times the dose can be administered without causing an intolerable reaction, local or general.

While at present vaccine therapy is not perfected, yet it exerts a colossal influence in a large percentage of the cases.

[Cases illustrative of knee-joint infections have been published in the CLINICS for April, 1912, pp. 177–180; December, 1912, pp. 825–832 (metastatic gonorrheal); August, 1913, pp. 637–646; October, 1913, pp. 831–843.]

[The above talk on autosensitized vaccines is supplementary to that by Dr. Kreuscher, which was published in the CLINICS for December, 1914, pp. 1119–1122. A fuller talk on vaccine and serum therapy appeared in the CLINICS for August, 1913, pp. 539–574.]

ANCIENT INFECTION OF HIP-JOINT: SECONDARY FLEXION-CONTRACTURE OF KNEE-JOINT FROM BURROWING ABSCESSES IN THIGH MUSCLES—OPERATIVE CORRECTION—TENOPLASTY

Summary: Clinical history of patient; comments—deformity of leg from neglect of treatment; two methods of overcoming this deformity; orientation of skiagram difficult; operation—avoid incising flexure-sites of body; division of contractures about knee; lengthening of biceps tendon; reposition of tibia into position of extension effected; extensor made of biceps muscle; recapitulation; plaster dressing—avoidance of pressure upon peroneal nerve; the old-time nerve-stretching for sciatica and for tetanus; postoperative comments; additional note—danger of traumatic endarteritis obliterans after operation; artery within nerve an important factor in maintaining a disturbed circulation.

HISTORY

The patient, a male aged thirty-one years, was admitted to hospital December 5, 1914. The family history revealed that two uncles and one aunt had died of tuberculosis.

The patient had had the usual diseases of childhood. Over three years ago, in the spring of 1911, he contracted gonorrhea, which lasted sixteen months, but which was not followed by complications. Eleven months ago, in January, 1914, there was a second attack of gonorrhea, which lasted three months.

The present trouble began twenty-three years ago, in May, 1892, when the patient was eight years of age. Pain developed in the right hip and lasted several days. One evening, hoping to obtain relief from the pain, he lay down behind the stove and fell asleep. Upon awakening, he got up and went out-of-doors.

He noticed no further trouble with the hip until several weeks later, on May 30, 1892, when he went in swimming in cold water. While in swimming, there developed severe pains in the right hip. He emerged from the water, dressed, and started to walk home. After walking about half a mile, the pain became so severe that he was compelled to sit down upon a sawdust-pile to rest. After the pain had abated somewhat, he continued his journey, and after covering three-quarters of a mile more, the pain compelled him to rest several hours before going home. Not only was the hip painful, but the patient limped. Arrived home, he went to bed and had a fair night. The next afternoon he went to bed again with severe pain in the right hip. Toward nightfall he got up and hobbled downstairs and went out for a while. During the next few days the hip was very painful.

About the latter part of June, 1892, the patient noticed a swelling of the right thigh, but says he had no chill or fever. On July 1, 1892, the right thigh was lanced in the region of the saphenous opening, and a great deal of pus was evacuated. Thereafter, sinuses appeared at various places in the thigh and continued discharging pus and pieces of bone for twelve years.

When he was twenty years of age (eleven years ago), the bone was curetted, whereupon the lesion healed and has never caused trouble since. While the sinuses were discharging, the thigh was very sensitive to touch. Simultaneously with the abscesses in the thigh the patient developed others on the right arm and in the lumbar region of the spine.

These abscesses and sinuses followed within a year after the onset of the trouble, and the patient was in bed during that time —from 1892 to 1893. He kept the leg flexed to ease the pain.

At present the patient is unable to straighten the knee. Upon walking there is full motion at the hip in every direction except that of complete extension and that of external rotation. He is unable to extend the right leg properly. There is moderate muscular atrophy in the right lower extremity. The patient has never been jaundiced and has never had a chill or fever.

COMMENTS

Dr. Murphy (December 9, 1914): As it looks to us, the story is one of secondary osteomyelitis in the upper end of the femur, and not the story of tuberculosis of the hip-joint. The original lesion was entirely in the hip. As a sequence of the in-

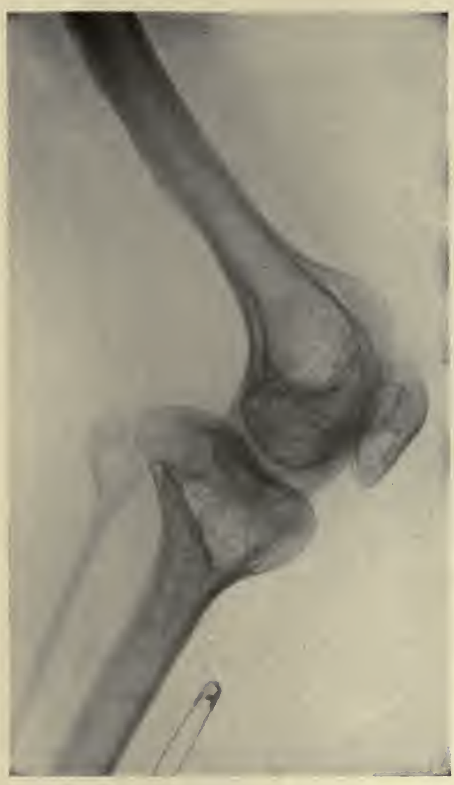

Fig. 243.—Preoperative skiagram showing secondary flexion-contracture of knee-joint. Shows degree of flexion. Note how contracting biceps has swung fibula around posteriorly (lateral view, see Fig. 245).

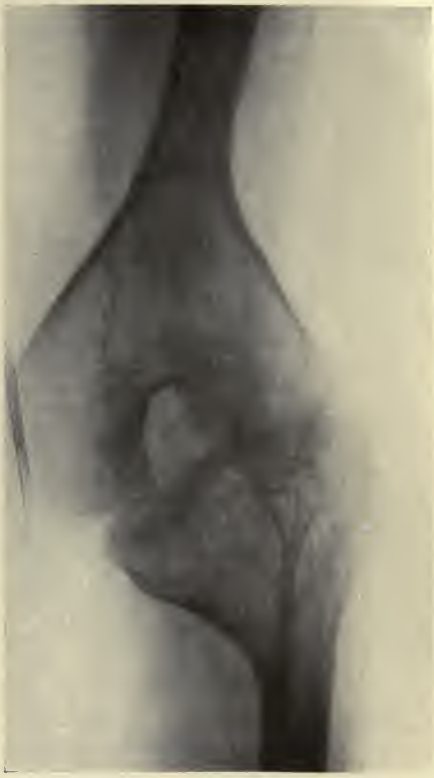

Fig. 244.—Preoperative skiagram showing secondary flexion-contracture of knee-joint. Contraction of the biceps femoris for twenty-three years has luxated and rotated the leg, so that in place of being square to the femur, the tibia is rotated outward into the position formerly occupied by the fibula, the fibula being drawn posteriorly (anteroposterior view, see Fig. 246).

flammatory process and of its neglected treatment, the leg was permitted to flex itself by degrees upon the thigh (Fig. 243), although there was at no time any disease in the knee. Because the disease was permitted to run riot twenty-three years without steps having been taken even to oppose the contraction of the scar tissue from the sinuses in the biceps muscles, the leg has become, in its flexed position, shorter than its fellow. Here are the openings of the sinuses; they are outlets of pus that gravitated along or in the biceps muscle, and they converted that muscle into a mass of cicatricial tissue. Continual contraction of the scarred biceps muscle during the twenty-three years' progress of the disease has resulted in subluxation and rotation of the leg, so that in place of being square to the femur, the tibia is rotated outward and clear around into the position normally occupied by the fibula, the fibula being drawn posteriorly (Figs. 245, 246, 247).

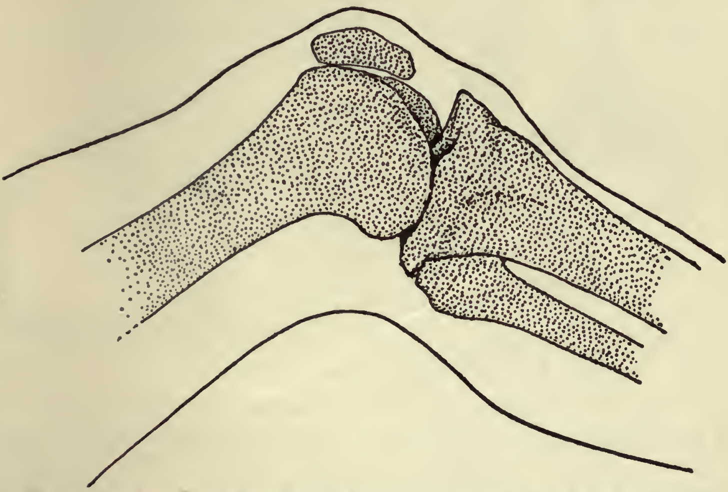

Fig. 245.—Sketch showing limit of voluntary extension of knee-joint. Note degree of external rotation of tibia on femur from long-standing contraction of biceps muscle.

Whether I can swing the leg around into place is problematic. He still has power in all the muscles of the leg. We can try one of two expedients: the first is to elongate all the internal and external hamstring tendons, and then see how far around we can bring the leg. If we can bring it far enough around so that he can stand upon it, the extensors will resume their control of the knee, and we shall not have to perform secondary resection. If we do not succeed in bringing it all the way, we shall bring it around as far as we can to-day and retain it there for a number of weeks, and then perform resection of the joint and make a straight, stiff leg in place of a leg that in the present position is useless. It is unfortunate that the neglect of treatment has driven us to such procedures.

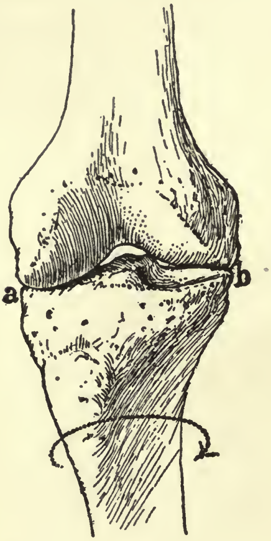

Fig. 246.—Sketch showing degree of rotation of tibia upon the femur by contraction of biceps. Anterior border of external tuberosity is on same frontal plane as posterior limit of external condyle. *a*, External, *b*, internal, aspect of joint. Arrow indicates direction of rotation in effecting reduction.

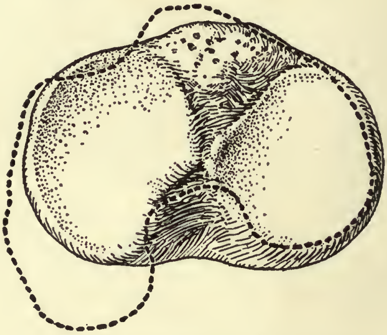

Fig. 247.—Schematic sketch showing relation of articular surfaces of femur and of tibia to each other. The tibia is rotated externally 40 degrees upon the femur. The internal condyle of the femur still rests in the hollow of the internal tuberosity of the tibia, but the *lowest* portion of the external condyle is anterior to the external tuberosity. (Femur shown in dotted outline.)

I shall demonstrate the relationship of the biceps tendon, showing you just how the tendon, by its long-continued contraction, has pulled the head of the fibula and with it the external tuberosity of the tibia clear around posteriorly in external rotation into the popliteal space (Figs. 246, 247). The tendon of the biceps is attached to the upper portion of the fibula, and by the muscle contracting to the superlative degree, its leverage upon the fibula has been so altered that the leg has been rotated until the foot became turned outward at a right angle with the long axis of the limb, and until it became impossible to extend the leg. When you look at that anteroposterior skiagram you would hardly know how to figure upon it. On the right side the picture is a very deceptive one, because of the distorted relations (Fig. 244). The stereo-skiagrams are much more valuable guides in planning the operation than the ordinary skiagrams.

The moment I try to bring the leg forward, external rotation of the leg results from the resistance offered by the contracted biceps tendon.

OPERATION

In making the incision, avoid injuring the external popliteal nerve; it lies internal to the tendon of the biceps, and with care may readily be avoided (Fig. 249). I shall make my arched incision and throw the flap backward. One endeavors never to make an incision in the popliteal fold. Incisions in the popliteal fold, incisions in the fold of the elbow, incisions in the anterior fold of the ankle, incisions in the anterior fold of the wrist, and incisions upon the palmar surface of the hand are always dangerous, owing to the subsequent contraction and the formation of keloids.

There is the biceps tendon: while I know the nerve is to its inner side, yet I do not know just how far down to find the nerve because this knee is rotated. There is the muscular portion of the biceps: I am freeing all these muscle-fibers from the structures behind, so as to relax the muscle. I am dividing one-half of the biceps tendon high up preparatory to its elongation, and shall split it downward to the head of the fibula (Fig. 248). There is the external popliteal nerve (Fig. 249): just watch the foot move when we irritate the nerve. The limb is no longer rotated because I have freed it from the clutch of the biceps tendon.

We next divide the strong external lateral ligament and the iliotibial band and let them recede (Fig. 249). There is the gastrocnemius muscle: you can see it contract with the exciter. We test the gastrocnemius with the Faradic current purely as a precautionary measure, to prove that we have recognized it and that it is still functioning. There is the posterior portion of the fibrous layer of the capsule: I am slowly stretching it. I must also release the fibrous origin of the gastrocnemius to facilitate extension of the leg (Fig. 249). As soon as you get the fibrous attachments loosened, then the muscles give. I am now leaving the muscles and throwing the divided end of the biceps tendon forward, preparatory to suturing it to the quadriceps extensor tendon, so as to convert the biceps from a flexor into an extensor.

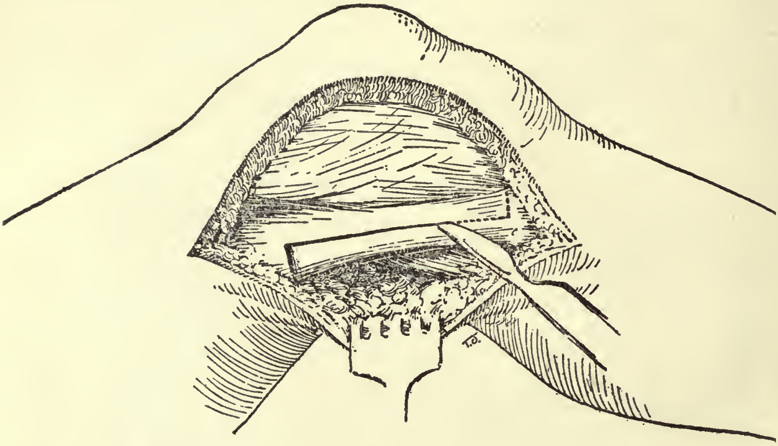

Fig. 248.—Sketch showing plan of splitting tendon of biceps femoris muscle preparatory to its elongation. The knife enters at the posterior border of the tendon close to the musculotendinous junction, and four inches above the fibula. After passing transversely through the posterior half of the tendon, the knife changes its course and passes vertically downward until it reaches the head of the fibula, where it changes its direction again and passes transversely forward, separating the anterior half of the bicipital insertion from the fibula.

One of the dangers associated with applying extension in this case is that of the great tension to which the external popliteal nerve will be subjected. Now we have succeeded in rotating the leg inward and restoring it to its normal relation with the femoral condyles: we have it straight at last. The next step is to suture the biceps tendon to the quadriceps extensor muscle (Fig. 250).

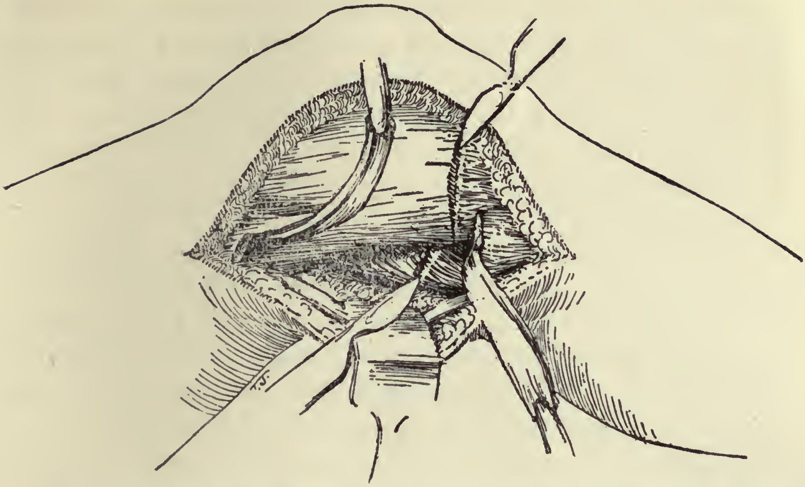

Fig. 249.—Sketch showing division of external lateral ligament and iliotibial band. The biceps tendon has been severed, and its ends drawn aside. The lower scalpel is seen dividing the outer head of the gastrocnemius muscle. The external popliteal nerve is seen freed and retracted posteriorly.

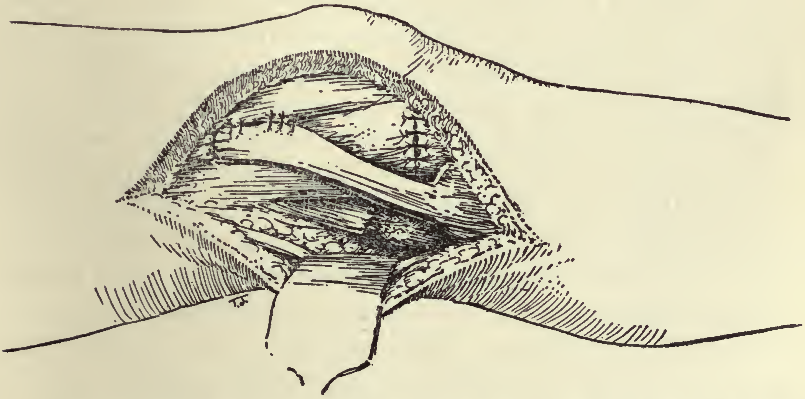

Fig. 250.—Sketch showing divided ends of elongated tendon of biceps femoris muscle stitched to each other and to the quadriceps extensor muscle, thereby converting the biceps from a flexor into an extensor. After division of the outer head of the gastrocnemius muscle the leg could be fully extended, and the external tuberosity of the tibia rotated internally into place. Cut edges of iliotibial band and of external lateral ligament sutured together.

I shall not have to do anything with the internal hamstrings: their unimpaired leverage will be of assistance in maintaining the leg *in situ.* I shall fashion a new external lateral ligament.

It is a question whether the popliteal circulation is going to be taxed too much by the tension to which it is now subjected, and whether the nerve, too, will be. The nerve, I think, will adjust itself to the tension.

Let the record show that the biceps tendon was elongated, and that an extensor will be made of it instead of a flexor.

Now you see I am going to convert the biceps muscle from a flexor into an extensor. The patient has enough power of flexion of the knee already. We are pushing the biceps tendon forward to implant it into the quadriceps tendon (Fig. 250). We make the same implantation when dealing with paralysis of the quadriceps following anterior poliomyelitis. Here is the vastus externus, which also aids in holding the tibia up in the position I desire.

RECAPITULATION

Let the record show that there is no bleeding; that we first exposed the tendon of the biceps at its attachment to the fibula and dissected it upward for four inches, freeing it from the structures behind; that we then located the external popliteal nerve and elongated the tendinous portion of the biceps by dividing it for one-half of its diameter at a site four inches above its insertion into the fibula, and then splitting the tendon downward and severing one-half of it at the fibular attachment (Fig. 248); that we then freed the aponeurotic and fibrous portion of the gastrocnemius and kept dividing it as the leg was extended (Fig. 249); that we then divided completely the femoral and fibular ligaments—that is, the external ligaments of the knee-joint—and freed the posterior portion of the capsule and the outer portion as far forward as the tubercle of the tibia; that with these tissues freed, traction with internal rotation was made slowly, and that without having to divide the internal hamstrings, we were able to slide the external tuberosity of the tibia along the inferior surface of the external condyle of the femur until its normal relation with the condyle was restored; that when this was accomplished it required but little pressure to make complete extension with slight adduction of the leg, whereas formerly the leg was in flexion with abduction; that so complete was the reduction of the deformity that the leg was practically straight and the subluxation of the tibia entirely overcome (Fig. 250); that on straightening the leg there was much relaxation of the quadriceps tendon, due to the fact that the leg had been in this superlative degree of flexion so long that the patellar tendon had become elongated; that we believe that the quadriceps muscle will contract sufficiently without our having to plicate or shorten the tendon subsequently; and finally that the leg will be dressed in a plaster-of-Paris case without extension.

Most of these muscles contract fairly rapidly.

Let the record show further that all the work was accomplished without hand-contacting and without opening the synovial membrane of the knee-joint, signifying that we have not exposed the knee to the hazard of infection.

DRESSING

Now we shall slowly stretch the leg, after the plan of Lorenz, until we restore it to its completely extended position. We feel the gastrocnemius giving, little by little. I am adjusting this plaster bandage in such a manner that it will not interfere with any swelling that may take place beneath it. Now the knee comes exactly where we want it—forward. There should be comparatively little swelling because there was comparatively little trauma, although an accurate dissection was performed.

We are cutting out a piece of plaster so as to spread the gypsum case where the external popliteal nerve comes around the neck of the fibula, because at that crossing you can get paralysis from two sources: one source is the extensive traction to which we exposed the external popliteal nerve, and the internal popliteal as well, for that matter. These nerves may be stretched and temporarily paralyzed, and yet function will return. To justify this statement, I need but remind you of the days when we used to stretch nerves for sciatica and for tetanus: I lived in the period when the treatment for tetanus consisted in stretching these nerves. We used to take a No. 28 male sound, put it under the nerve, and saw and saw with it, and the wonder is that any of the axons were left and had retained their potency of regeneration. But some of the axons must have been left, or else permanent paralysis would have resulted.

POSTOPERATIVE COMMENTS

Now the patient has a leg upon which he can walk for the first time in twenty-three years. He may have foot-drop from the stretching of the nerves, just as patients used to come out of an anesthetic with wrist-drop, from the weight of the arm resting upon the operating-table for an hour, or an hour and a half. That was known as chloroform-paralysis, but it was not chloroform-paralysis—it was table-pressure paralysis.

The toes are pretty pale: if they stay pale we shall move the leg little by little every day until the circulation adjusts itself, because, owing to the patient's youth, we know that the artery itself is strong and healthy. The artery may stretch, but it should not break at his age. Were he sixty years of age, we would have arteriosclerosis and its attendant evils to contend with. We shall elevate the leg upon three or four pillows and not let the bedding rest upon the foot. Just as soon as he awakens we shall urge him to send impulses through these limb muscles, because their contraction favors the circulation.

There can be circulation through the nerve even though the arterial circulation to the limb be cut off. I recall a case in which the femoral artery had been ligated, and yet the total circulation through the peroneal nerve was as great as that in the radial artery, the collateral circulation going right through between the bundles of the nerve and supplying the required amount of blood.

We did not apply a circular bandage to the leg, but a "barber-pole" bandage, so that there is no constriction such as there would be from a circular bandage.

ADDITIONAL NOTES

Dr. Murphy (December 10, 1914): Regarding the case of tenoplasty we operated upon yesterday, I wish to state that in the early part of the afternoon the limb was cold and quite anemic; later in the afternoon it began to improve, however, and last night the circulation was good. The patient has, on the other hand, complete suppression of motion in the territory supplied by the peroneal nerve, indicating that the axons have been stretched, so that there is no transmission of impulses through the nerve below the site of stretching. He is able to tell that he is being touched; that, I believe, is due to the fact that it is the territory of the anterior crural nerve that is being tested, and not really of the peroneal. In the posterior part of the knee sensation is very feeble.

The patient has been mentally prepared for the probability of amputation as one of the hazards to which he would be exposed by our attempt to obtain extension of the leg. The liability of amputation still clouds his convalescence, because of the danger of endarteritis obliterans of the traumatic type developing from the stretching of the vessels and the resulting tension. Will the artery thrombose? If the artery thromboses suddenly, he will get gangrene of the leg below the knee; if it thromboses gradually, he will not get gangrene. Often the artery that runs through the nerve between its bundles is a wonderful help in maintaining the circulation during the interval between the time of suppression of the circulation and that of its reëstablishment by means of an outside collateral circulation.

[*N. B.*—December 11, 1914: The circulation in the foot is excellent. The patient still has complete paralysis of the leg, due to the stretching of the nerves at operation.]

The circulation was gradually restored. The nerve function was completely restored by the end of twenty days. He has good motion in his knee, and practically full voluntary extension of the leg.

[A case in which the tibia was subluxated backward, and in which resection was performed with a view to securing ankylosis in extension, was published in the CLINICS for August, 1913, pp. 611–616. Dr. Hochrein's skiagrams, illustrating the blood-supply to the knee, which are referred to in this article, are shown in the same issue, pp. 589 and 591.]

TUBERCULOSIS OF KNEE-JOINT—RESECTION BY THE AUTHOR'S CONCAVOCONVEX METHOD

Summary: Clinical history of patient; comments—differential diagnosis, tuberculosis, Charcot joint—illustrative cases; operation—active tuberculous process contraindicates arthroplasty; pathologic picture tallies with skiagram; concavoconvex resection performed; avoid inserting a foreign body in the presence of tuberculosis; recapitulation; treatment of tuberculous knee-joint of an adult *vs.* a child.

HISTORY

The patient, a male aged thirty-three years, was admitted to hospital September 5, 1914. The family history revealed that one sister died of pulmonary tuberculosis. The past history was negative.

The present condition began twelve years ago, in 1902, in a slight injury to the inner side of the left knee. The patient kicked a brace of the door with the left leg, kicking inwardly in the direction of the right leg. In this action he knocked the knee against the door. There was severe pain lasting for less than a minute, and then becoming insignificant. He could flex and extend the knee as before the injury and without pain.

Three or four weeks after the injury he noticed a swelling, uniform in shape, appear at about the site of the injury and gradually increase in size. There was no pain except when he stumbled or jumped upon the left foot. He had no chill preceding the appearance of the swelling and does not recall having had fever.

His condition continued about the same until three months later, when he noticed that the swelling was not diminishing and that the knee felt weaker, the weakness causing him to stumble at different intervals. Four months after the accident, while riding a bicycle, he noticed that, as the left pedal of the wheel went down, his foot did not follow it the whole way: it was evident that the knee could not be bent as much as before. He still had no pain.

Four years after the injury he could not ride his bicycle on account of inability to flex and extend the leg. At that time he began to have fever. He avoided bearing the usual weight upon the left leg, and used a cane occasionally.

For the last six months the degree of motion has been greatly restricted. He has pain if the usual body-weight be borne upon the foot. Grating has been present in the knee for the past six years.

The patient states that, after resting in a chair, his knee often seems as though it were caught when he starts to rise. In order to overcome this catching he has to shift the knee at different angles. The pain is increased if he tries to flex the knee to any extent. He has gained 15 pounds in weight during the past six months.

COMMENTS

Dr. Murphy (September 11, 1914): Here are the skiagrams of this case. The lateral view shows practically nothing (Fig. 251). The anteroposterior view (Fig. 252) shows that the external condyle is excavated, or gnawed out. You will note in looking at these pictures that the one in the left side of the shadow-box shows an irregular mottling of the lower end of the femur and of the upper end of the tibia, but when you come to look at the picture to the right (Fig. 252), on the left side of it you will notice that, in addition to the involvement of the proximal portion of the external condyle of the femur, the external tuberosity of the tibia has been absorbed, the external tuberosity being from one-half to three-fourths inch lower than the internal tuberosity, indicating the destruction of a considerable portion of the external tuberosity of the tibia.

But the inspection of the skiagrams does not tell you what is the matter with the patient—the history tells you what is really the matter. He went to kick a brace of the door and developed pain of such severity that he fell down and was immediately incapacitated momentarily. In a few days the whole trouble

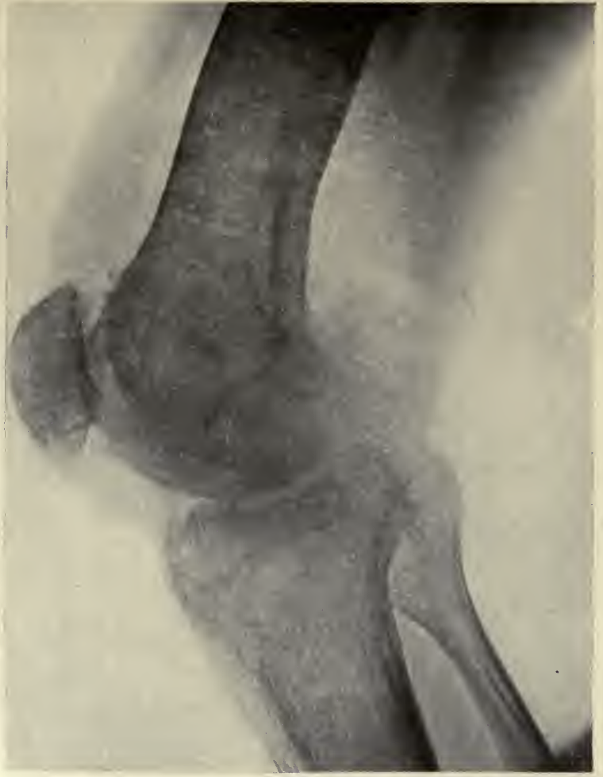

Fig. 251.—Preoperative skiagram of tuberculous knee-joint (lateral view). The pathologic features are shown better in the anteroposterior view (see Fig. 252).

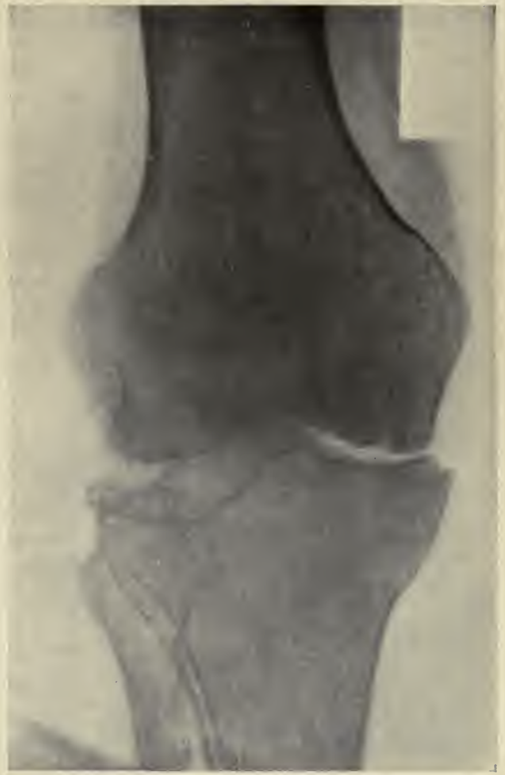

Fig. 252.—Preoperative skiagram of tuberculous knee-joint (anteroposterior view). Note excavation or gnawing out of proximal portion of external condyle of femur. Note also absorption and dropping of external tuberosity of tibia, which is from one-half to three-fourths inch lower than the internal tuberosity.

passed away, and he felt perfectly well. Three or four weeks later the knee began to swell. Three months later the swelling was still present, and the knee felt weaker, giving way at intervals. Motion became more and more restricted until, finally, he could not use the limb. There were periods of exacerbation and improvement, but the improvement was never sufficient to repair fully the damage done by the last attack. There was fever when the motion began to be restricted. He has not lost weight, but on the contrary has gained 15 pounds within the last six months.

The story makes the diagnosis, and infection of a joint, of any other origin than tuberculous, incapacitates the patient within forty-eight hours. A joint-tuberculosis never incapacitates an adult before the end of three and one-half or four weeks. That is the rule. Occasionally in a Charcot joint you have the same manifestations, from the changes that occur in the joint in connection with syphilis of the spinal cord. These Charcot joints, however, do not have the severe pain at the time of the initial trauma; the changes in the joint come on often as rapidly as they do with tuberculosis, and are as progressive as they are in tuberculosis, but they are not accompanied by the primary or secondary severe pain.

Let me give you an illustration of the "story" of a Charcot joint. A man was pushing a wheelbarrow filled with earth over a plank, when suddenly his left foot slipped off of the plank, resulting in a sprained ankle. He was able to finish the day's work, however, and to ride his bicycle home. The pain was not sufficient to incapacitate him, and he continued working every day. Two weeks after the injury, on getting out of bed one morning, he noticed that the left hip was swollen, but that *there was no pain.* At the end of five weeks he consulted a physician, who, because of the large swelling at the hip-joint, diagnosed sarcoma of the femur initiated by the trauma, and then sent him to me. I examined him and concurred in the diagnosis. He turned to go out of my office. I said to him: "Why do you limp? does it hurt you?" He said: "No, it has never hurt me; it did not hurt me when I fell, even." "Hold on a minute: It did not hurt you when you fell?" "No." *That* told the story—that he had a Charcot joint. (See CLINICS, February, 1912, p. 64.) No pain with the initial trauma—a trauma severe enough to fracture a leg caused no pain! *There is practically no pain in a Charcot joint, nor in a Charcot fracture.* We found that he had the Argyll-Robertson pupil, and that he had also the Romberg sign and absence of patellar reflexes—*he* had no sarcoma. This shows you how easily I was misled by not paying sufficient attention to the clinical manifestation of pain after the injury.

Another man came in with a very badly deformed ankle-joint. He had sustained an injury to the joint and was treated for a Pott's fracture; he bore malice against his physician because the result was extremely bad. I looked at the ankle and found the foot turned in. A foot is never turned in as a result of a Pott's fracture with deformity, but is always turned out. That was proposition No. 1. I asked him how much pain he had. He replied: "I have never had any pain." "How about when you broke it?" "I have never had any pain at any time." The skiagram showed rarefaction and absorption of the lower end of the tibia and of much of the astragalus. The only other sign of tabes he has was the Argyll-Robertson pupil. He had a typical Charcot ankle for which his physician was wholly irresponsible. (See CLINICS, April, 1912, p. 153.) I explained the situation to him and told him that he should go and explain to those to whom he talked and apologize to his physician, and that if he wished, he might tell them that he had had syphilis when a boy, and that this was the sequence. I do not think he carried out the latter instruction, but his case shows you with what caution you must analyze these cases from a clinical standpoint.

Such is the clinical course of joint-infections, and their incidence is so uniform in each type of infection that it would save us many errors if we would devote close attention to the matter of the clinical course of the disease. It should be supported by all the laboratory findings that one can bring in as aids in the diagnosis.

As a result of the tuberculosis never having been fully repaired, this patient has exacerbations of the disease. You might recall the case we presented about ten days ago, in which the man had trouble in a joint for a number of years, and then an acute attack. We sent an excised specimen of tissue to the laboratory, and the pathologist, knowing nothing of the clinical course, reported its being incipient tuberculosis with an acute exacerbation. That was also the final report from the laboratory and showed an acute as well as an old lesion.

OPERATION

Now this knee-joint has not undergone changes beyond which it is impossible to perform arthroplasty, provided, of course, that there be no active process in the joint. Therefore, I am going to plan my incision on the basis that I shall be able to perform arthroplasty if there be no active tuberculosis. Never perform arthroplasty in the presence of active tuberculosis, else a serious situation arises. We are not using iodin on the skin in the neighborhood of this, an arthroplasty-site, because it sometimes produces chemical dermatitis, which retards repair and which might be a source of danger.

[Goblet-shaped incision made (Fig. 253).] I am incising this quadriceps tendon through the *external* half of its area above the patella (Fig. 254, *A* to *C*). After splitting in the midline the quadriceps tendon, the patella, and the ligamentum patellæ (Fig. 254, *A* to *A′*), I incise the ligamentum patellæ through the *internal* half of its area below the patella (Fig. 254, *A′* to *B*), so that we can retain that patella after splitting it through the middle.

Note the bloody fluid in the joint and the villus formation. There is the tuberculous process in its activity, and below is the *brie* material. The synovial membrane is wholly destroyed, and the activity of the process means that I cannot perform arthroplasty.

Now we shall adapt our incision for a resection. I have made a full exposure of that field, and the synovial membrane must come out. There is the ligamentum patellæ: witness the tuberculosis attacking the patella. This picture shows how nature tries to repair the destruction wrought by tuberculosis—how she protects and defends and finally is overcome by the disease. The

SERIES OF SKETCHES ILLUSTRATING THE STEPS OF DR. MURPHY'S OPERATION OF CONCAVOCONVEX RESECTION OF KNEE-JOINT

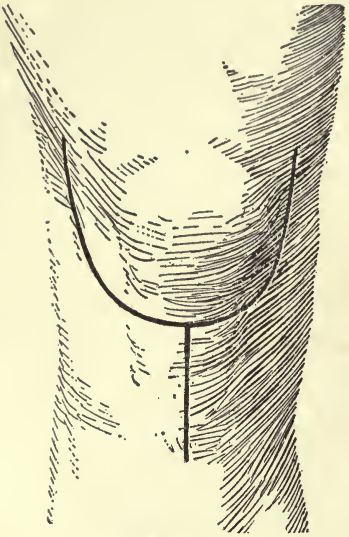

Fig. 253.

Fig. 253.—Shows goblet-shaped incision of skin as the first step in the concavoconvex resection. This incision is also the best for exploratory operations upon the knee-joint, since it affords the freest exposure of the deep fascia and circumpatellar aponeurosis (anterior view).

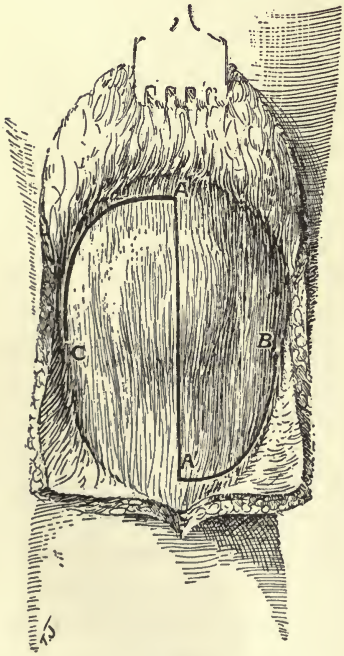

Fig. 254.

Fig. 254.—Shows reflection of skin-flaps. The upper flap (the cup of the goblet) is turned upward, and the lower flaps (the stem of the goblet) are turned to each side. The field thus developed comprises the quadriceps tendon above, the patella in the center, and the ligamentum patellæ below, while on each side the lateral expansions of the quadriceps aponeurosis are seen. These tissues are incised as follows:

A to *A′* represents the median incision, and splits from above downward in the midline the quadriceps tendon, the patella, and the ligamentum patellæ.

A to *C* represents the curved superior external limb, and divides the external half of the quadriceps tendon and the aponeurosis and then sheers off vertically to stop at the top of the external tuberosity of the tibia.

A′ to *B* represents the curved inferior internal limb, and divides the internal half of the ligamentum patellæ and the quadricipital aponeurosis, and then sheers off vertically to stop at the top of the internal tuberosity of the tibia, opposite the termination of the external limb (anterior view).

subquadriceps bursa is destroyed and obliterated, and must be excised. Here is the quadriceps tendon of the patella. Having planned to perform arthroplasty, I have split the patella through the middle, but later shall just approximate its halves.

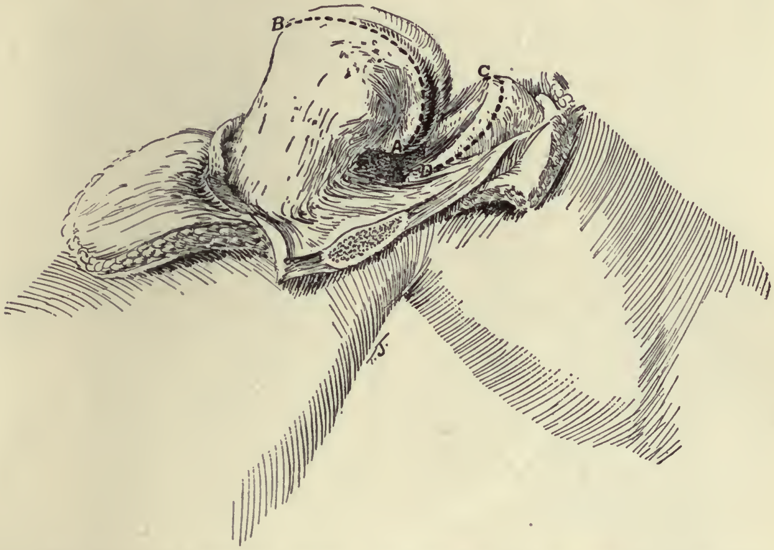

Fig. 255.—The osseo-aponeurotic flaps outlined in the previous figure have been freed and reflected to each side, exposing the articular ends of the femur and tibia. The leg has been partially flexed upon the thigh, exposing the whole of the inferior articular extremity of the femur. The concavoconvex resection of the bone-ends is made as follows:

A to *B* represents the convex line for the femur followed by the Gigli saw, which, adjusted at *A*, follows the convexity of the articular surface of the femur as it travels forward just beneath the articular cartilage. When the sliver of bone is removed, it will be seen that the convexity of the condyles has been preserved (concavo*convex* resection). *C* to *D* represents the concave line for the tibia followed by the Gigli saw. The sliver of bone is removed in similar manner, but upon its removal the upper surface of the tibia is left concave (*concavo*convex resection) or moon-shaped. Both the convex and the concave saw lines should have the same arc, so that when the leg is extended the convexity of the femur will fit into the concavity of the tibia (see Fig. 256) (lateral view).

Now I am freeing a portion of the fibrous capsule that I am going to retain. The next step is the reconstruction of the bony surfaces. Formerly we sawed these surfaces *square* off, and as a result had very poor bony apposition if the angle of the leg

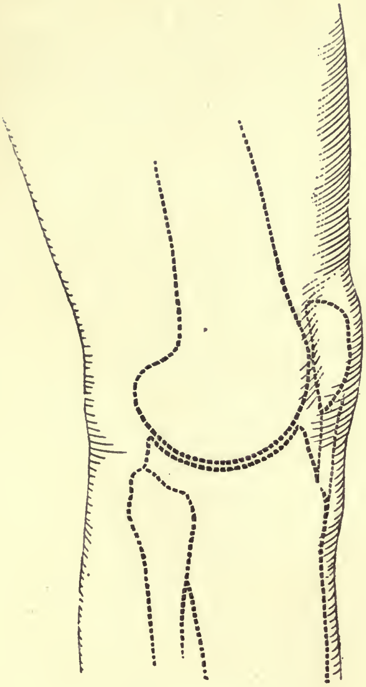

Fig. 256.—Schema of Dr. Murphy's method of concavoconvex resection of the knee-joint. The convexly prepared end of the femur rests in the moon-shaped depression that was fashioned in the upper end of the tibia. Regardless of the angle of flexion or of extension of the tibia upon the femur, a constant large area of bony contact is maintained, so that anterior or posterior displacement of the tibia is prevented. To obtain such accurate contact it is essential that both the convex and the concave saw-lines have the same arc (see preceding figure) (profile view, orientating bones that compose skeleton of knee-joint with the surface).

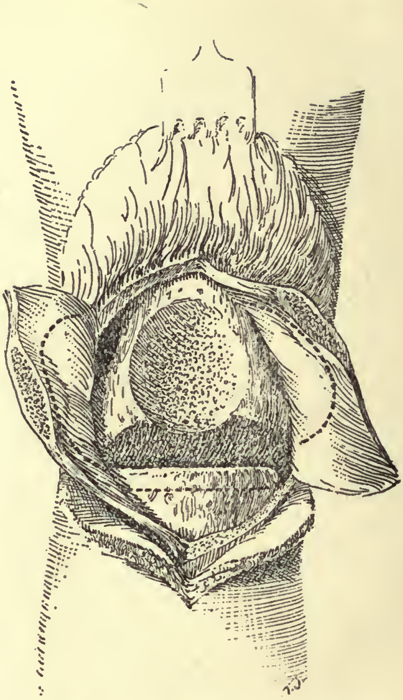

Fig. 257.—Sketch showing method of dealing with patella when, after concavoconvex resection of the knee-joint in a young subject, and after the tuberculous process has been entirely eliminated, one intends to reëstablish motion subsequently by performing arthroplasty.

The lateral osseo-aponeurotic flaps have been reflected, and on the under surface of each the dotted line shows the outline of the flap of capsular fascia and fat which will be reflected over the patellar fossa of the femur to prevent the patella from organically uniting to the freshened surface of the femur.

The dotted line on the tibia represents the lowermost level reached by the convexly prepared surface of the femur. The limb is in a straight position (anterior view).

became altered. In those days we used for fixation catgut, phosphor-bronze wire, nails, and all sorts of mechanical agents. Now we employ the concavoconvex method of resection which was devised by us (Figs. 255 and 256).

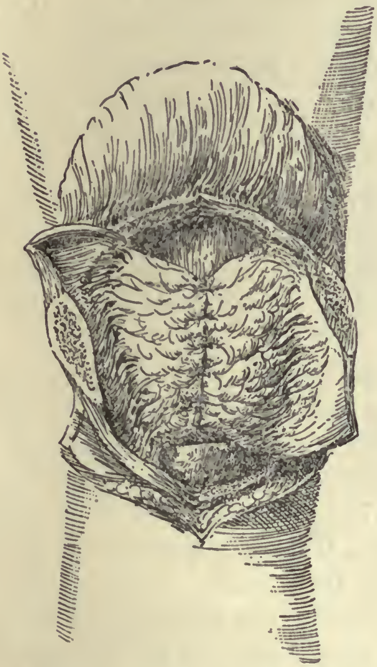

Fig. 258.—Sketch showing lateral flaps of capsular fascia and fat elevated by dissection, brought across the freshened patellar fossa of the femur, and stitched together so as to insure against subsequent bony ankylosis of the patella to the femur. This step is taken immediately after concavoconvex resection of the knee-joint when one intends to mobilize the knee-joint later, as mentioned above (anterior view).

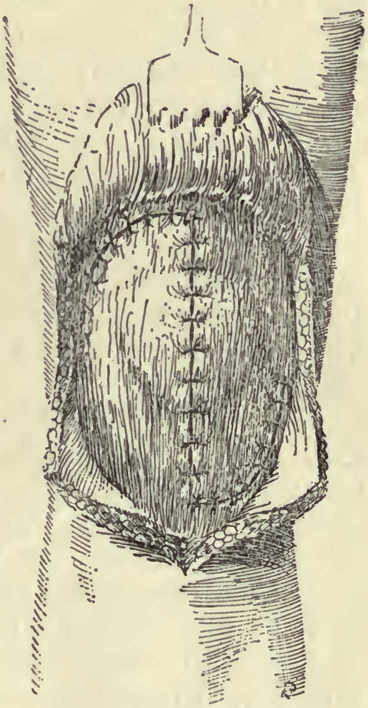

Fig. 259.—Sketch showing reapproximation of edges of quadricipital aponeurosis and tendon of patella, and of ligamentum patellæ. The more the quadriceps muscle contracts, the more firmly will the cut edges of the patella be contacted, just as after a vertical incision into the rectus abdominis muscle (anterior view).

See that tuberculous *brie!* It is in a large pocket of the synovial capsule back of the external tuberosity of the tibia, at the place where the skiagram showed the greatest amount of destruction (Fig. 252). You can see how truly the skiagram portrayed the lesion. We shall be able to make a curved resection of only one of the condyles of the femur. Note how I adjust the Gigli saw at the upper limit of the cartilage, covering the condyle posteriorly, and saw downward and forward, keeping just beneath the cartilage, and following the convexity of the condyle (Fig. 255, *A* to *B*). Now, you see, I am changing the angle of the saw as I advance so as to make this rounding-off of the condyle V-shaped. Below, there is but one tuberosity that is suitable for resection.

I did not run the saw across the posterior part of the joint because occasionally with a cicatricial contraction the vessels are attached close upon the bone. I recently read of a surgeon who performed one of these operations and had, as a sequel, gangrene of the leg.

Above, I shall leave all the bone that I possibly can because there is such a destruction of the external tuberosity of the tibia that I shall be obliged to take off a considerable portion of the upper end of the internal tuberosity. Now we shall resect the upper end of the tibia; I shall have to fashion a moon-shaped depression of its upper end so that the convexly prepared end of the femur may rest upon it (Fig. 256).

The fibrous capsule is never the seat of a primary infection of any kind. The infection begins in the bone or in the subsynovial vascular membrane. Now you can see me pick out that subsynovial membrane, situated deep in the operative field well back of the external tuberosity. It incloses a large pocket of *brie*. See how nature has defended it by inclosing it within the fibrous capsule, constantly striving, by her constructive process, to build up a defense and heal the diseased bone: there is the encapsulated mass. That is the type of tuberculous process that reacts to the tuberculin test with a low temperature and a slight constitutional reaction. Why? Because it is excluded by a coffer-dammed area.

That pathologic picture corresponds with the skiagram to a nicety: note how the latter reveals the extensive area of destruction in the external tuberosity. What a great aid skiagrams are when we properly interpret them, and how disastrous it might be when they are improperly interpreted!

We use phenol in wiping out the joint, just as it was used originally, forty years ago. Now we suture the ligaments with catgut, not with wire: *never insert a foreign body in the presence of tuberculosis!* See the reason for making that concavoconvex resection? I have nothing to support this condyle. I have a splendid support on the other side, so that the condyle will remain in that position. Now we shall suture the quadriceps tendon: you see what it does by its tension—forces these two bony surfaces into accurate adjustment all the time and at all angles and insures union (Fig. 256). That is a most important factor after the removal of the disease—the immobilization of the structures that formerly were mobile.

RECAPITULATION

Let the record show that this knee must be dressed in an adducted position so as to throw all the pressure upon the internal condyle, and that the foot must be left at a right angle with the leg.

Let the record show further that the external tuberosity was destroyed for a depth of more than one-half inch; that the tuberculous process in the tuberosity was encapsulated and was enucleated in its tuberculous capsule, but that an active tuberculosis was present elsewhere in the subsynovial layer of the capsule; that in the synovial capsule, fairly well back of the external tuberosity, there was a large pocket of *brie*, and that this pocket and sac were enucleated *in toto;* and finally that we performed our operation of concavoconvex resection of the knee-joint.

[NOTE.—There was primary union, and in eleven weeks there was good, firm, bony union with the limb in a straight position.]

DR. MURPHY: Once a tuberculosis is established in a knee-joint of an adult, it is practically hopeless of repair with any type of expectant or non-operative treatment. Rest, extension, injections, etc., do not offer sufficient hope of reward to justify the expenditure of time and the suffering which they entail. Primary excision, with a production of arthrodesis of the concavoconvex type within a few months after the onset, is the proper line of treatment.

If the patient is young, the patella should be retained and prepared with a view to performing a subsequent arthroplasty to reëstablish motion after the tuberculous process has been entirely eliminated (Figs. 257 and 258).

[Cases of tuberculosis of the knee-joint have been published previously in the Clinics for August, 1912, pp. 607–619; February, 1913, pp. 83–98.]

PAINFUL STUMPS OF LEGS—REAMPUTATION—EXCISION OF NEUROMATA—NEURORRHAPHY

Summary: Clinical history of patient; comments—histology of repair and degeneration of a divided nerve; importance of glial repair; proper disposal of a nerve-stump; Thoma's theory of tropism of cells; catheter-cystitis and pyelitis avoidable by massaging bladder for retention of urine; operation—proper disposal of periosteum in resecting the bone-ends; neuromata found and excised; recapitulation.

HISTORY

The patient, a male aged thirty-five years, was readmitted to hospital in April, 1915, suffering from pain and tenderness in the amputation-stumps of both legs.

Over a year ago, in March, 1914, both legs were amputated in the lower third in a railroad accident. The stumps were trimmed up elsewhere. Since healing, the stumps have felt cold and are very sensitive to pressure, even the weight of the sheet being intolerable. In the same accident he suffered a fracture-luxation of the right hip-joint, which was reduced by operation in this hospital on September 12, 1914.

COMMENTS

Dr. Murphy (April 27, 1915): This patient suffered traumatic amputation of both legs in the lower third, and with the same accident there occurred fracture-luxation of the head of the right femur. This hip lesion was not recognized at the time of the operation, performed elsewhere, when amputation stumps were trimmed up, in March, 1914. When he came in here six months after the accident the luxation could not be reduced manually, or after exposure, so that we had to excise the head of the femur and insert the neck into the acetabulum. He has now a wide range of motion in the hip-joint. Previous to our operation he could not use the luxated limb owing to the great adduction-deformity.

In dealing with the trunks of large nerves it is very important to consider the histology of the repair and regeneration of a divided nerve. At first, when a nerve is cut, the axons have no contractile power. Finally, as scar tissue forms, the nerve contracts, and the glial tissue which surrounds the axons contracts and forces the axons to stick out like brush-ends, and these can be seen with a pocket-lens. The glial tissue is the connective tissue of the nervous system, and differs from ordinary connective tissue in its ectoblastic origin.

As repair progresses in the nerve it contracts, and on the end of the nerve there forms a bulb which is termed a neuroma—a postoperative neuroma. What happens next? The same phenomenon occurs that follows the repair of connective tissue elsewhere in the body: the scar-tissue consolidates and contracts, thereby compressing the sensitive axons. How can we prevent the occurrence of neuromata? By dividing the nerve in such a manner that it will heal by another process than by this repair through ordinary connective-tissue capping. All the repair must be made by means of the proliferation of glial tissue in the large axons.

Glial repair is established thus. The nerve-end is not cut squarely across, but it is cut across transversely for one-half of its diameter at a point three-fourths of an inch from its end: this cut half is split vertically downward to the end of the nerve, and is then removed. The distal end is treated in the same manner. These two prepared rectangular ends are then contacted and stitched together. By this method of treating the nerve-ends there is nothing more than a glial tissue covering, and a neuroma does not form. When dealing with a single nerve-end, as in this case, the nerve is prepared in the same manner, and the end is folded back upon the nerve and stitched to the heel of the bayonet-shaped nerve-end.

Axons have a great affinity for each other. Thoma was the first to call attention to the tropism, or affinity, or positive chemotactic attraction between cells of the same histologic structure.

It was demonstrated that these axons will penetrate ordinary loose connective tissue for some distance to meet each other. When a nerve has been cut and contacted with a muscle that runs parallel to its long axis, the axon will span a still greater distance. If there be a mass of connective tissue one-fourth, one-sixth, or even one-thirty-second of an inch thick between the ends, non-union will result. This interposition of fibrous tissue, which forms an impenetrable barrier across the path of the growing axons, explains so many failures of union.

In reviewing the history of nerve-repair, one finds that the profession was divided into two great schools. The members of one school contended that if nerve-ends were sutured together, function would be restored; those of the other school claimed, however, that, under these conditions, function is not restored. When I was a student I remember the late Dr. Gunn's having had to deal with a divided musculospiral nerve. He found the nerve-ends, approximated them with sutures, and secured ultimately functionating union. That was one of the very early examples of neurorrhaphy; but I think the reason why Dr. Gunn secured functionating union was because he operated before the stage of connective-tissue capping. That the members of the other school did not succeed in restoring function was due to their sewing the nerve-ends together without first having freshened them: it were as logical as to sew two pieces of cheese together and expect them to unite.

Eleven years ago, in 1904, we analyzed the clinical course and results in hundreds of cases of nerve-suturing. We found that if the nerve-ends be freshened until the fasciculi are seen, and then the fasciculi are contacted, union with restoration of motion is just as inevitable as in a recent fracture that has been accurately reduced and contacted. On the other hand, union with restoration of transmission is never obtained unless the nerve-ends be trimmed until the healthy, brush-like axons have been exposed. A simile might be drawn between the two nerve-ends and the two ends of a cable. In uniting two lengths of an electric cable it is not enough merely to contact the leaded ends: these leaded ends must first be trimmed off, and the individual wires exposed and contacted, otherwise no transmission of electric impulses will occur.

In addition to his painful stumps this patient has a catheter-cystitis with pyelitis. That is, he has an inflammation that began in the urinary bladder from undue catheterization, and extended upward through the ureter to the kidney. Catheter-infection of the kidney usually results from meddlesome surgery. Retention of urine may be overcome by massaging the bladder *via* the rectum in the male, and *per vaginam* in the female.

OPERATION

[Incision and exposure of interior of stump on right side.] In resecting the bone-ends I must leave the fibula long, because the tibia is very short; usually, however, it is better to resect the fibula at a slightly higher level than the tibia. While formerly we preserved the periosteum and covered the bone-ends with it, now we do the opposite, resecting the bone-ends from one-half to one inch *below* the level of the removal of the periosteum, thereby preventing postoperative pain.

There is a nerve: it is the posterior tibial. I lift it up and remove the fibrous cap: see the brush-ends of the axons sticking out, exactly as I described them! This nerve has been inflamed, and appears very large. The tropism or positive chemotaxis of which I spoke will attract these axons toward each other. We shall deal with this nerve-end by trimming it so as to obtain glial union, according to the method described above. This nerve was drawn out at the time of the previous operation. Here is the anterior tibial nerve: it has developed what I described to you—a neuroma which is as large as an olive. This neuroma explains the patient's inability to wear an artificial limb. Is not that pretty? The development of the neuroma shows what happens when the nerve-end is not dealt with as above. To prevent neuroma-formation, just turn the bared axons toward each other, and nature will do the rest. That is the key to the situation.

[The stump of the left leg was treated in the same manner.]

[Additional information on the subject of neurorrhaphy may be found in the following numbers of the Clinics: February, 1912, pp. 24–30 (illustrations of neuroma, of brush-like axons, and of method of nerve-lengthening); April, 1914, pp. 355–368 (amputation-neuroma with ascending neuritis—division of right half of cauda equina); February, 1915, pp. 77–92 (division of brachial plexus, with illustration of a cluster of neuromata on p. 87); August, 1915, pp. 667–675 (section of musculospiral nerve in fracture of humerus); see also the next case.]

[Note.—Patient was immediately relieved of pain.]

PAINFUL STUMP OF LEG—REAMPUTATION—EXCISION OF NEUROMATA—NEURORRHAPHY. THROMBOANGIITIS OBLITERANS

Summary: Clinical history of patient; comments—review of history; operation—preliminary subcutaneous tenotomy of biceps femoris; trophic and ulcerative changes in stump; reamputation—nerve-ends treated so as to prevent neuroma-formation; postoperative comments—progress of thromboangiitis obliterans; involvement of digestive organs and of renal arteries in that disease; case of plugging of both renal arteries with necropsy findings; thromboangiitis obliterans confined almost exclusively to males of the Jewish race.

HISTORY

The patient, a male aged thirty-four years, a Hebrew, was admitted to hospital May 10, 1915. The family history is negative. The past history shows that nine years ago, in 1906, the patient had an attack of gonorrhea which lasted three months.

The present malady began sixteen months ago, in January, 1914. While the patient was out driving in sub-zero temperature, the right foot began to feel cold. He took off the shoe and noticed that the foot had turned pale. After rubbing it a little he drew on the shoe and drove the remaining two miles to town. He took a drink of spirituous liquor, and then went home and immersed the foot in hot water. A few hours later the foot began to swell, and became red and painful. He went to work the next day, but the foot ached and was very painful by night. The foot remained painful continually.

Two months later, in March, 1914, he noticed three purplish spots appear upon the great toe. He consulted a physician, who said that the great toe should be amputated. This amputation was performed on April 28, 1914. Two weeks later the edge of the foot turned black and blue, and the leg was amputated above the ankle. Immediately following the amputation the patient noticed throbbing pain in the stump, and this pain continued three weeks, and then the stump became tender to pressure, and has remained tender since.

Six months after the second amputation the patient put on an artificial leg, but could not endure it on account of the pain in the stump. The right knee became stiff while he was in bed from his keeping the knee constantly flexed. At present the leg is in a position of acute flexion, and the patient cannot extend it beyond a right angle.

Before the foot was frozen, in January, 1914, there had been no symptoms of aching or of pains in the legs severe enough to make him stop or sit down and rest, nor had there been anything to suggest intermittent claudication. Between the time of chilling of the foot and that of amputation, however, he had attacks of pain when on the feet, and was forced to sit down and rest during the attacks.

Examination by Dr. Murphy.—The patient has thromboangiitis obliterans in the right leg, with the claudication which so commonly is associated with that malady.

COMMENTS

Dr. Murphy (May 12, 1915): Previous to the time of amputation the patient could not walk three blocks to save his life. He had typical intermittent claudication from the time of the chilling of the right foot to that of amputation. He chilled the right foot and it became pale; the circulation in the foot reestablished itself, but upon exercising, the foot became purplish-blue. After harder exercise it became pale.

The patient is a Hebrew and shows the typical and classic condition of thromboangiitis obliterans, which occurs only in members of his own race: I have never seen this disease in any other race. The sequence is first chilling of the foot, then gangrene, and finally amputation.

OPERATION

I may have to perform subcutaneous tenotomy in order to straighten the leg. This operation is performed in this case by inserting the tenotome beneath the skin and dividing the tendon with the tenotome. This division takes the tension off the external hamstring entirely. The hamstrings consist externally of but one muscle, the biceps femoris, while internally there are three muscles—the semimembranosus, the semitendinosus, and the gracilis, and, if you wish, a part of the sartorius. The leg can now be extended: I do not know whether we shall be able to keep it extended, but we shall try. This straightening of the limb relaxes the capsule in front and results in contraction of the gastrocnemius muscle. Now we must proceed with the reamputation and find the nerves.

Let the record show that the trophic changes in this stump have been extreme, and that not only was the nerve trophic, but the stump ulcerated, so that we have this ulceration, in addition, to contend with.

Now we can resect about two inches more of this stump and still have control for the artificial leg. We resect a little more of the fibula than of the tibia. In resecting the tibia always round off its prominent anterior corner so it will not press against the skin of the flap and cause ulceration.

Let the record show that the bone was much softer than normal.

See the axon-ends of the nerve stick out like brush-ends! That is the continuation of the external popliteal nerve: here is the other branch. I want to prepare this nerve-stump for suture. The thing we wish to prevent in this case is recurrence of the neuritis. I am splitting this nerve-end and turning one-half of the split end back upon itself, after removing the other half of the split end: otherwise a neuroma will develop on the end. This suture is passed through the end of the nerve, securing it in its folded condition.

Here is the other nerve. I shall likewise stitch the ends of its axons together by splitting the nerve-end and plicating it back upon itself so that axon contacts with axon. By this procedure we lessen the liability of neuroma-formation, as well as of post-operative neuritis. The important problem in this case is the treatment of the nerve-stumps. This nerve-end is of much greater size than we usually conceive of. We have an accurate approximation of these axons now, and a neuroma should not develop, nor should the stump be sensitive any longer. If the neuritis from which this patient has suffered be not checked, it will travel higher and higher until finally we shall be obliged to cut into the cauda and there divide the nerves in order to relieve the pain, as in a case we reported previously (Clinics, April, 1914, pp. 335–368).

Now I shall show you how I treated the nerve-end. Let us suppose that this hand represents the nerve. You cut transversely half way through the nerve three-fourths inch from its end, and then split it down to the end and remove the incised half. Then you trim the remaining end and fold it over upon itself until the end contacts with the proximal end of the cut half at the heel of the bayonet-shaped nerve-end, and secure it in that position by stitches. Thus, the axons of the nerve-end become circuited. If you let the nerve-end remain exposed in the open, it will eventually be covered over with a connective-tissue cap and, as a sequence, a neuroma will form. Owing to the diseased state of the vessels, there was practically no bleeding: no constrictor was used. [Closes flap.]

POSTOPERATIVE COMMENTS

Thromboangiitis obliterans first manifests itself by the patient's inability to walk any distance without having to sit down and rest. Preceding this inability to walk—by months and even years—there is the development of flat-foot, which is so typical in these cases. After the inability to walk there appears claudication, followed by complete occlusion of the blood-vessels, and this eventuates in gangrene. In your desperate efforts to check the progress of the disease you amputate first one leg and then the other leg, but despite your efforts the gangrene creeps slowly and slowly upward until it involves the back of the leg: it rarely, however, extends above the knee. "Claudication" of the stomach and of other digestive organs as well may appear. A splendid article was written upon this subject seven years ago and contained a large number of collected cases (Buerger: *Amer. Jour. Med. Sci.*, 1908, n. s., cxxxvi, 567).

Thromboangiitis obliterans may involve one or both renal arteries. When it involves a renal artery, the patient has a typical attack of renal colic. I remember one of my early cases in which both the patient's legs had been amputated. The patient was admitted later to the Michael Reese Hospital, suffering from a typical attack of renal colic—pain lasting several days and then disappearing, and followed by blood in the urine. He was treated for renal colic by three different physicians, and finally came under my observation in the Presbyterian Hospital, where he was admitted on Dr. Sippy's service. Four days after admission I saw him in the throes of an attack of apparent renal colic, and with complete suppression of urine. I believed the anuria of the other kidney to be reflex in origin, and of the type which Dr. Bevan described once as the sequence of a calculus in the ureter. We decided to operate upon the left kidney—the side upon which the pain occurred. We exposed the left kidney and decorticated it: *I cut it, but it did not bleed!* It was as dead as Julius Cæsar—not a drop of blood in it. In its progress the thromboangiitis obliterans had affected the renal artery not only of this left side, but also of the right side. The patient lived one week without voiding one drop of urine. On the seventh day he died, and autopsy revealed complete plugging of both renal arteries. This case illustrates the fact that renal manifestations *do* occur in thromboangiitis obliterans, and are due to involvement of the renal arteries in that disease.

Thromboangiitis obliterans is classic, almost always confined to the Jewish race, and almost always to males: I have never seen it occur in a woman. This patient is only thirty-four years of age, and yet there is complete obliteration of the vessels of the right leg. The disease will develop upon the other side later, probably involving the renal arteries and terminating fatally.

POTT'S FRACTURE WITH EVERSION DEFORMITY: NON-UNION OF TIBIAL MALLEOLUS, UNITED FRACTURE OF FIBULAR MALLEOLUS, AND POSTERIOR LUXATION OF ANKLE — OPERATIVE REDUCTION OF DEFORMITY

Summary: Clinical history of patient; comments—demonstration of skiagram; difficulty of this class of cases; preparation of skin of patient; operation—fracture of both malleoli with non-union of tibial malleolus and posterior luxation of ankle; recapitulation; postoperative comments—difficulties of reduction.

HISTORY

The patient, a female aged thirty years, was admitted to hospital December 25, 1914. The family and past histories were negative.

The accident which caused the present trouble was received two years ago—in 1912. The patient stepped backward into a hole, sustaining a fracture of the ankle. In the accident the foot was everted until the inner side of the ankle touched the ground. There was immediately severe pain in the ankle, and she was unable to place the foot upon the ground. The foot became swollen and ecchymotic. A physician reduced the fracture and applied a wire splint, which included the whole leg: this splint was retained ten days. Upon removal of the wire splint a plaster-of-Paris dressing, which extended up to the knee, was applied and retained three weeks, during which time the patient was up and about on crutches. She walked with the aid of crutches for six months after the gypsum case had been removed.

At the present time the patient is lame, the foot being slightly everted. She has pain when walking.

COMMENTS

Dr. Murphy (December 26, 1914): On looking at the skiagram you see first that the tibial malleolus is displaced outward half way across the articular surface of the tibia; secondly, that there is a gap between the fibula and the tibia filled with bone; thirdly, that there is a displacement of the fibula and of the tibial malleolus backward, and a displacement forward of the rest of the tibia; and finally that the astragalus is three-fourths of an inch farther back than it should be.

There is no operation that we dread or dislike more than the very class of cases of which this is an example. In the first instance, such cases are mechanically difficult; secondly, even when reduced, there is difficulty in maintaining reduction; thirdly, there is great danger of subjecting the posterior tibial artery to tension, with subsequent gangrene of the foot. For two weeks after the operation one is in a state of great anxiety about this complication developing, because sometimes things occur over which we have no actual control. We recall such a case, in which there resulted gangrene of the great toe back to the interphalangeal joint, and gangrene of the tip of the second toe.

The first question to arise is that of amputation. We never try to reduce the deformity now without first informing the patient of the risks of the case—how the circulation might be shut off by tension thrust upon the arteries, and how gangrene might arise, necessitating amputation of the leg above the ankle. That explanation is put down in writing in the presence of the patient, but while it saves one from future entanglements with the patient, yet it does not relieve him of anxiety. Notwithstanding these difficulties and dangers, our results have been good with the exception of the patient who lost the great toe.

We have two cases of this kind in hospital now, in one of which we had to resect the posterior portion of the articular surface of the tibia in order to be able to bring the astragalus forward. The patient is a soldier in the United States army, and he would have been permanently out of commission unless we had done something for him. He already has good motion, and is going to have a good result. The other patient will also have a good result.

I want to call to your attention the posterior luxation of the astragalus: you can see that the tibia is situated anterior to the astragalus. That backward luxation is hard to overcome, because you can now see that as soon as you reduce it you at once bring tension to bear upon the posterior tibial artery.

We do not, as a rule, use iodin, particularly upon thin-skinned patients, but the operative field is so extensive here that it cannot be covered with towels.

OPERATION

[Applies constrictor and makes incision.] There you see a cavity in the lower end of the tibia: the tip of my instrument goes right into it. It seems to me that the fibula did not have bony union. The posterior half of the fibular malleolus appears to be intact, but the upper, anterior half appears to be broken off. We shall cover the field with towels. There you see the tibia sticking out: its anterior margin projects above the astragalus and three-fourths of an inch anterior to it. I must go into the joint and find where the tip of the tibial malleolus is. You see it beneath my finger: I can push it downward; it was not united at all, but had fallen half way across the lower articular surface of the tibia, and was displaced backward and outward. You can see that, as I manipulate it, it comes back into position, and that I have freed it entirely from the tibia.

Now we shall go to the outer side. There is the posterior margin of the fibular malleolus, and I want to call your attention to the way in which it has been split. There is the line between the tibia and the fibula. This anterior portion of the fibula has been broken off; the posterior portion has been displaced upward and behind the articular surface of the astragalus, as is indicated in the skiagram.

There is the astragalus: here the displaced tibia. It is the horizontal articular surface of the tibia which has remained intact, while the tibial malleolus has been carried backward with the fibula, just as the skiagram reveals. There is the line of union. There is the hollow space that shows in the skiagram. The tibial malleolus is united above and below, and is free in the middle. There is the fibular malleolus, three-fourths of an inch behind its normal site.

Now we must resect the anterior portion of the tibia in order to restore the bone to its normal relationship with the astragalus, as well as enough of the fibula on the outer side to permit the fibular malleolus to come forward and into place. The astragalus has been displaced back much farther than usual. . . Those who are near can see how I have corrected the posterior displacement of the astragalus, and I must now secure the fibular malleolus with a screw, so as to support and retain the astragalus (Figs. 260 and 261). [Removes constrictor.]

RECAPITULATION

Let the record show that it was extremely difficult to effect the reduction; that the fibula was freed from its posterior attachment, swung around forward, and was secured with a screw to aid in the fixation of the astragalus and to prevent a recurrence of the deformity; that after the removal of the constrictor there was practically no bleeding, so that no ligatures had to be used; that the lower end of the tibia had to be resected in order to effect reduction and to obtain a comparatively flat surface upon which the astragalus could rest; and finally that all the work was accomplished without hand-contacting.

POSTOPERATIVE COMMENTS

You saw the great difficulty which we encountered in overcoming the backward displacement: this same difficulty has been met with in all the cases of this type.

We shall sterilize all this exposed operative field with iodin. You must watch the state of the circulation in the foot every day: if the circulation should fail, you must swing the foot around out of place again. [Applies gypsum case.]

Let the record show that after the application of the gypsum case the circulation in the toes and in the foot was perfect, and

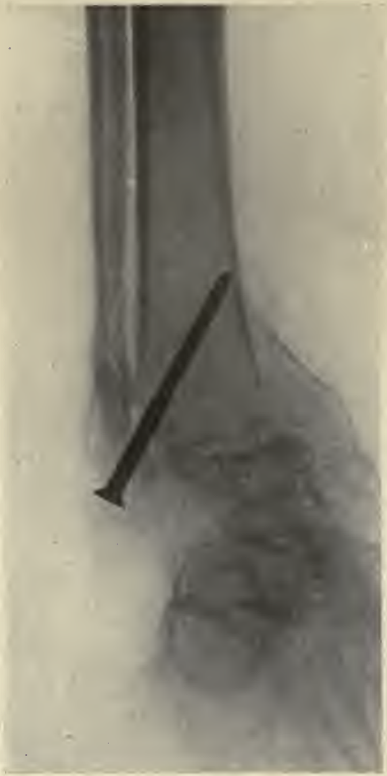

Fig. 260.—Postoperative skiagram showing fracture of fibular malleolus. Secured by a screw driven through it and into the tibia, thereby retaining the astragalus in place. Note inward displacement of tibial malleolus (anteroposterior view).

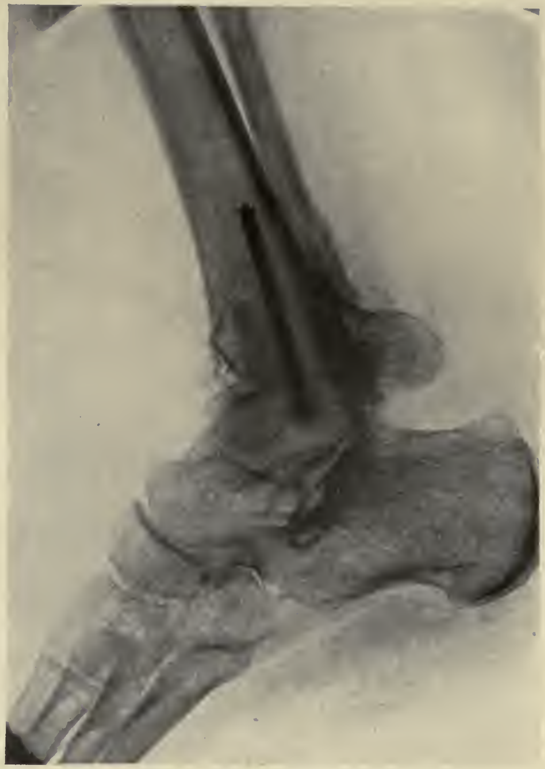

Fig. 261.—Postoperative skiagram showing screw that retains fibular malleolus *in situ* (lateral view).

that we removed a window from the gypsum case to facilitate after-dressings.

In the treatment of Pott's fracture one has to take a great many details into consideration. Next to a Colles's fracture a Pott's fracture yields the greatest percentage of poor results—not poor because the proper splint is not applied, but because it is difficult to effect the reduction and maintain it. It is not so difficult to reduce a Pott's as a Colles's fracture, but when one sees a Pott's fracture there is the natural tendency to bring the foot forward in attempts at reduction—that is, by making dorsal flexion; the fact is, however, that to effect the reduction one must make plantar flexion, extending the foot as he brings it down instead of trying to dress it in that position [indicating], which is impossible.

[*N. B.*—The patient made an uneventful recovery. She left the hospital about the tenth of February, six weeks after operation, and at that time already had good motion in the ankle-joint.]

[Cases of Pott's fracture have been published in the CLINICS as follows: June, 1912, pp. 451–457 (10 illustrations); August, 1912, pp. 618–621 (Students' Clinic); February, 1914, pp. 1–27 (14 illustrations); October, 1914, pp. 993–1001 (in the same number, pp. 967–992, there are two cases of fracture (*not* Pott's fracture) by *inversion*), and in the August (1913) number, pp. 593–595, are shown Dr. Hochrein's skiagrams of the arterial supply about the ankle, the importance of preserving the integrity of which has been referred to in the above case.]

1026

RICHMOND STEPHENS, M. D.

THE CLINICS
OF
JOHN B. MURPHY*
Volume IV Number 6

LEUKOPLAKIC PAPILLOMA OF BUCCAL MUCOSA—ABLATION OF PAPILLOMA—PLASTIC FLAP RESTORATION OF MUCOSA

Summary: Clinical history of patient; comments on leukoplakia and leukoplakic carcinoma; surgery, and not radium, offers most in the therapeusis of leukoplakic carcinoma; operation—leukoplakic papillomatous patch excised, together with tissues down to periosteum, and including the latter; flaps of mucosa elevated and sutured over raw surfaces; recapitulation; wounds in mouth heal rapidly.

CLINICAL HISTORY OF PATIENT

The patient, a male aged fifty-seven years, was admitted to hospital May 26, 1915. The family history revealed that his father died of carcinoma of the stomach, and a sister of tuberculosis. The past history is negative.

About fourteen months ago, on March 14, 1914, the gum bordering upon the patient's lower second molar tooth began to be sore, and soon the tooth itself became painful and remained painful for three days. A small abscess formed along the gum line and opened itself spontaneously, with relief from the pain. After the opening of the abscess the patient noticed for the first time that the involved gum and the mucous membrane of the cheek were whitish in color: he does not know whether or not there was an ulcerated or whitish area along the gum or buccal mucous membrane before the tooth became sore—in fact, he noticed nothing wrong until he felt the toothache. A dentist lanced the abscess along the gum-line and drilled into one of the roots of the lower second molar tooth: he found the nerve in the root dead and the tooth loose. When the dentist drilled into the tooth, the patient had no pain. The dentist cleaned out the root-canal and treated the tooth for one week, during which time the patient had no pain. In order to gain better drainage of the abscess cavity, the dentist eventually drew the tooth.

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* This number of the Clinics was edited by Dr. P. G. Skillern, Jr., of Philadelphia.

He then treated the ulcer left remaining after extraction for one year, but could not make it heal. A physician finally advised the patient that the gum-lesion was cancer.

Examination by Dr. Murphy.—The leukoplakic papillomatous patch extends from the socket of the extracted left lower second molar tooth over the fauces to the mucosa of the outer part of the soft palate (Fig. 262). It is of the buccal type, and does not bleed, even when handled: it has burrowed deep into the tissues. The lower tooth in front of the involved area will have to be removed, and the deep cervical lymph-nodes will have to be ablated.

COMMENTS

Dr. Murphy (May 28, 1915): The patient has a leukoplakic papillomatous patch, and when in a position where it is subject to trauma, as in this case, such a lesion is always a source of danger.

The etiology of leukoplakia is not clear, and the malady is most obstinate to treat. Chromic acid in 1 per cent. solution and even osmic acid preparations have been used, but after their application the leukoplakic patches have still continued to thrive. When occurring at the buccal angle, these leukoplakic patches are extremely dangerous, and must be closely and constantly watched for the first clinical evidence of malignant degeneration. The infection of the tooth preceded the formation of this leukoplakic papilloma. These leukoplakic papillomata do not bleed; they send epithelial processes deep down into the mucous membrane; they begin by heaping up and displacement of the epithelial cells, the cells in many cases penetrating the basement membrane and then becoming malignant. Furthermore, leukoplakic carcinomata of the mucosa of the cheek or mouth form the most malignant of all types of carcinoma, with the exception of the melanotic type. They do not yield to x-ray treatment, nor do they yield to radium. The Radium Institute in London, of which Colonel French is in charge, will not accept a case of leukoplakic carcinoma for treatment at all. During my visit to the Institute I inquired, "What about your results in the

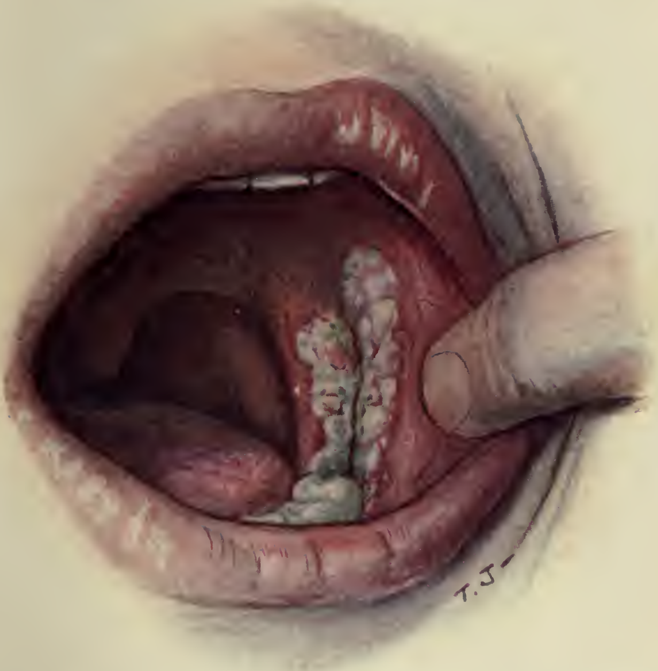

Fig. 262.—Sketch showing leukoplakic papillomatous patch extending from socket of extracted left lower second molar tooth over fauces to mucosa of soft palate. (Natural tints.)

treatment of leukoplakic carcinomata by radium?" Colonel French said, "We do not treat cases of leukoplakic carcinoma, since they do not yield to radium therapy."* Therefore, surgery —and surgery early—is the only method of treatment that promises these patients any relief at all.

Leukoplakic carcinoma cells do not metastasize to the lymph-nodes early, but the cells appear to be thrown out in clumps after the plan described by Handley, in which the cells are pushed away from the center. In describing the advance of carcinoma from the primary focus after penetration of the basement membrane, Handley says that they do not grow up by piling up on each other, but that they grow by piling up on the proximal end, and are forced forward by the lymphatics into the lymph-spaces well beyond the area involved. With that view we are not in accord. We are becoming more and more convinced that transmission of metastasizing malignant cells through the *blood*-stream is very common. In the statistics on postmortems of cancer cases, which we collected recently, we found that in carcinomata developing in sites below the level of the clavicle the cells metastasize most commonly into the lymph-nodes; next in frequency into the liver, and that the liver plays a second rôle in all types of metastases in cancer except in those situated above the clavicle. I recall one of my very early experiences in the case of a woman, about forty years of age, who suffered from carcinoma of the breast. There was no evidence of carcinoma in the lymph-nodes. Within a few months after we removed the breast she became icteric, showing metastases to the liver, in complete accord with cases incorporated in our statistics. The liver enlarged by degrees, but before the liver was enough involved to cause her death, metastatic carcinoma developed in the brain and caused lethal exitus.

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* Sticker (*Berl. klin. Woch.*, October 4, 1915, No. 40. Abstract in Jour. A. M. A., November 20, 1915, 1856) reports fifteen cases of cancer of the mouth in most of which the malignant disease retrogressed under radium treatment without scars or mutilation.

In three the cancer was on the tongue and the radium induced a clinical cure. In one case only seven twelve-hour exposures, all at night, accomplished the purpose, the cancerous crater healing completely in a few weeks. In seven cases the cancer was in the lower jaw and in five others in the upper jaw. The radium was fastened in a plate made to fit over the upper or lower teeth. One patient wore this for thirty nights' exposures, and it did not interfere with his sleep. In two other cases the cancer was a recurrence after one or several operations. By modifying the tooth-plate-holder it was possible to apply the rays in various directions, thus facilitating the cure.

OPERATION

There is the neoplasm: you can see here these leukoplakic papillomatous areas. At the upper angle you see the socket, from which the tooth was extracted yesterday. [Incision made around leukoplakic papillomatous patch.] We shall wait a moment until the oozing stops, so that we can go right down to the periosteum. [Excises leukoplakic patch, including the periosteum.] We have had enough teeth removed to give us an opportunity of covering the whole denuded area, so that it will be entirely free from trauma in the future. We shall control this oozing and then get ready to sew up. [Outlines two mucosa-flaps—one from mucosa on under surface of tongue, the other from uninvolved mucosa of cheek (Fig. 264).] You see with what facility I am enabled to bring the flap of mucosa obtained from the inner side of the mouth over the alveolar process and suture it to the flap of mucosa obtained from the cheek, thereby covering over this exposed area left remaining after ablation of the growth (Fig. 265): the employment of flaps is essential to free the mucosa from plication and essential to the prevention of the development of scar-tissue over the bone. We shall not ablate the lymph-nodes to-day because we have a fresh wound in the mouth, and that fresh wound can be the source of infection of the cervical field from which the lymph-node would have been removed. If you will look into the mouth, you will see how nicely the whole operative zone has been covered over.

RECAPITULATION

Let the record show that after removing this mucosa, which was not ulcerated, but which was papilliferous, I have removed all the tissues down to and including the periosteum of the mandible at the angle of the jaw, but did not remove any bone; that two flaps were prepared and freed—one from the mucosa on the under surface of the tongue, the other from the uninvolved mucosa of the cheek—to cover the mandible, and were shifted so as to cover all the raw surfaces, and then were sutured into place. Look! You should have no trouble in recognizing the sites from which the flaps were obtained, and that the exposed area is now entirely covered over by the flaps.

Wounds in the mouth heal very rapidly when aided by a mouth-wash of lysol or corrosive sublimate—the lysol is the more agreeable.

[*N. B.*—For further course see next case.]

RECURRENT LEUKOPLAKIC PAPILLOMA OF BUCCAL MUCOSA—ABLATION OF PAPILLOMA—PLASTIC FLAP RESTORATION OF MUCOSA

Summary: Clinical history of patient; comments—papilloma of buccal mucosa, while microscopically non-malignant, is, like Paget's disease of nipple, clinically malignant from the beginning in 100 per cent.; importance of early and thorough ablation of the tumor; no necessity of removing lymph-nodes of neck so long as cells of neoplasm have not penetrated mucous membrane; sites of metastasis from carcinoma of lip; operation—ablation of neoplasm and covering of denuded area with flaps from mucosa of tongue and of cheek; recapitulation; postoperative note.

CLINICAL HISTORY OF PATIENT

The patient, a male aged fifty-seven years, was readmitted to hospital September 22, 1915. Four months previously, on May 28, 1915, the patient was operated upon by Dr. Murphy (*vide supra*), and left hospital in June.

Since the operation the white, granular patch on the gum and buccal mucosa has not increased in size, and possibly has even become a little smaller; at any rate, the patient expected it to disappear entirely. There is no pain, nor any irritation; the patch is not tender.

COMMENTS

Dr. Murphy (September 23, 1915): The pathologist's report on the specimen removed at the previous operation was that it appeared to be non-malignant, fibro-epithelial in type, despite the excessive proliferation of the surface epithelial cells, which gave the growth its white, cartilaginous appearance. In other words, it was a mucosa-papilloma. All the papillomatous tissue was removed at the first operation. The involved area was not ulcerated, and did not bleed when we touched it, nor does it bleed now when touched. It was ablated just a few months ago—on May 28, 1915. The growth has recurred, and the pathologist reports that it is non-malignant. What does he mean by non-malignant? He means that while there is a colossal heaping-up of epithelial cells *above* the basement membrane, yet these epithelial cells have *not* penetrated to beneath the basement membrane, and, therefore, the neoplasm is microscopically non-malignant. Clinically, however, it is malignant, and such a mucosa-papilloma is malignant from the very first day that you see it. It is the story of Paget's disease of the nipple over again: the Paget nipple microscopically is non-malignant for many years, while clinically it is malignant from the very beginning in 100 per cent. The moment you see a papilloma like this one, occurring opposite the ramus of the mandible and appearing white, you may know that it is clinically malignant in 100 per cent. Unlike the leukoplakic patches, these papillomata grow up from the beginning as wart-like excrescences and harden from the trauma to which they are subjected during mastication, and during the contraction of the buccinator muscle externally. Eventually they always ulcerate; and eventually, always become malignant.

When this patient left here he was warned that if there appeared any evidence of return of the trouble he should come back to hospital immediately. After its removal I shall again send this specimen to the microscopist, and I know that he will again report that it is non-malignant, because it has not undergone ulceration, nor penetrated the mucosa. Starting at the angle of the mouth, the white mucosa-papilloma goes deeper and deeper into the submucous tissues, not ulcerating, but merely burrowing, and a similar state of affairs is seen in the burrowing of an infected trochanteric bursa, which, however, is non-malignant. [See CLINICS, April, 1913, pp. 337–344.]

This time I am planning to ablate a still wider extent of the involved area, and then to swing another pedicled flap of oral mucosa from the under surface of the tongue, and contact that sublingual flap with a similar flap from the uninvolved portion of the buccal mucosa. To insure ablation of the papilloma throughout its entire extent I intend to dissect as carefully as I can, and yet I operated radically once before upon this case, but the neoplasm recurred. If the papilloma continues to return, it will finally become malignant in the microscopic sense, and ultimately terminate the life of the patient. In ablating this type of growth it is not necessary to remove the regional lymph-nodes, because there has been no ulceration, nor any penetration of the basement membrane by epithelial cells.

The rapidity of return of this papilloma signifies that it is malignant in the clinical sense. Its return is accelerated by its occupying a site where it is subject to repeated trauma. If extensive ablation of the neoplasm wide of the tissue involved is performed early, and if there is no return in the lymph-nodes and no evidence of metastasis on palpation, then one may feel fairly sure of the total absence of metastatic involvement, even when dealing with a carcinoma. If, on the other hand, the ablation is performed late, then there is present the ordinary type of carcinoma that is so common at all sites in the body, but that differs entirely from, for example, carcinoma of the lip. Though the most exposed of all carcinomata, yet epithelioma of the lip is considered one of the most malignant of all malignant epithelial growths, and it gives rise to the most virulent metastases to regional lymph-nodes. The metastases from

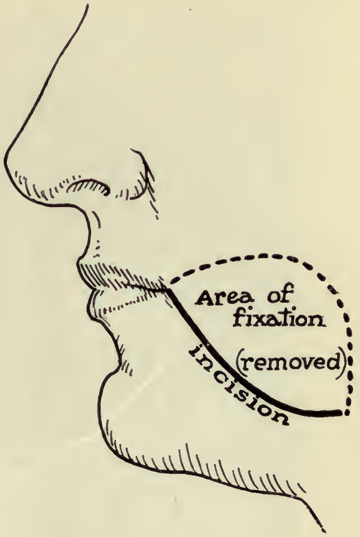

Fig. 263.—Shows line of incision through cheek. Between the heavy and the broken line there is shown the area of fixation of the cheek to the mandible. This and the two succeeding sketches were made at the operating table from a case similar to that described in the text, and serve to show the general plan of plastic flap operations to cover denuded areas in the mouth.

carcinoma of the lip do not commonly invade the bones, nor travel to the liver, but mostly to the lung and pleura. The time to remove these buccal or labial carcinomata is the first day they are seen by the physician. Do not temporize by daubing the growth with nitric acid, phenol, or glacial acetic acid, but excise completely and thoroughly the entire zone involved.

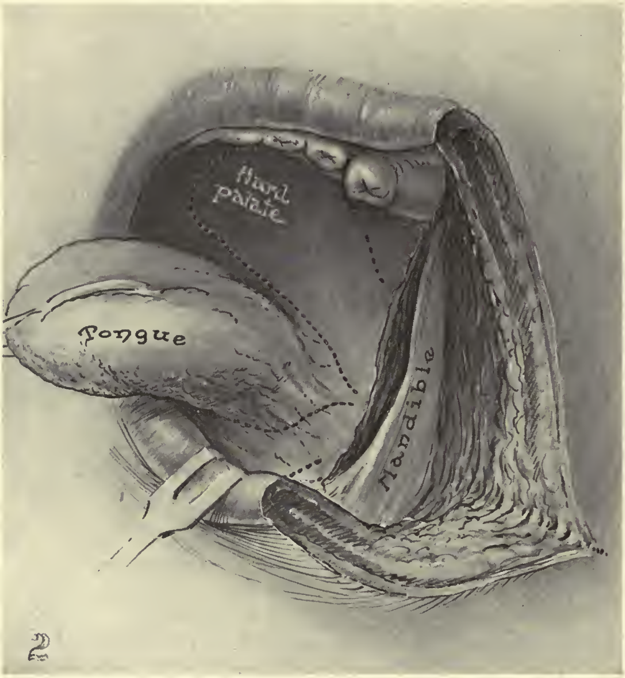

Fig. 264.—Shows bared ramus of mandible and adjacent denuded surfaces left remaining after ablation of the neoplasm. The flaps of mucosa—one from beneath the tongue and the other from the palate—are shown in dotted outline.

OPERATION

See that white patch back in the corner of the mouth? [Incision around area involved.] Note that I am going wide of the neoplasm into healthy tissue. . . Now I am right down upon the bone, taking off the periosteum with the tumor. . . I next fashion from the under surface of the tongue a flap of mucosa, and bring it forth into the field so as to cover the denuded area left remaining after ablation of the neoplasm (Fig. 264). Gauze pressure for a few moments will stop the oozing from the area uncovered by removal of the flap. . . In addition, I fashion a flap from the mucosa of the cheek, and suture it over the alveolar process to the mucosa-flap from the under surface of the tongue. If I do not cover the bared area with mucosa-flaps, the patient will have limitation of the movements of the mandible, if not complete ankylosis.

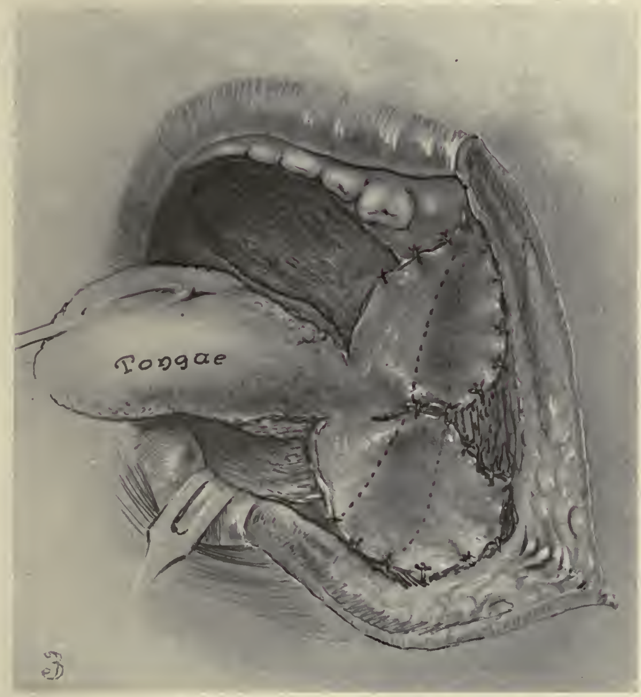

Fig. 265.—Shows flaps swung around on their pedicles and tacked into place. The position of the now-covered ramus of the mandible is shown in dotted outline.

RECAPITULATION

Let the record show that the neoplasm was removed together with the periosteum, exposing the alveolar process; that the denuded surface was covered in by two mucosa-flaps—one from the under surface of the tongue as far back as the circumvallate papillæ, and the other from the mucosa of the cheek; and that these flaps were sutured together over the exposed alveolar process.

POSTOPERATIVE NOTE

The patient left hospital October 4, 1915. The wound was entirely healed *per primam.* The mucosa-flap obtained from the tongue had united firmly with that obtained from the cheek.

PAPILLOMA OF LIP AND OF CHEEK—EXCISION—PLASTIC RECONSTRUCTION

Summary: Clinical history of patient; comments—diagnosis of lesion based upon physical examination; general scheme of sites of formation of papillomata within mouth; untreated papillomata eventually become malignant; malignant neoplasms of buccal mucosa the most malignant of all cancers and respond neither to radium nor to *x*-ray; operation—necessity of removing papillomata by going wide of the growth into healthy tissue and excising the entire base; removal of papilloma and reconstruction of lips.

CLINICAL HISTORY OF PATIENT

THE patient, a male aged forty-nine years, was admitted to hospital June 14, 1915. The family and past histories are negative.

Seventeen years previously, in 1898, the patient noticed that his throat had become sore. He consulted a physician, who diagnosed chancre of the tonsil. A section removed for microscopic examination verified this. Three months after the appearance of the chancre the patient noticed a patch about as large as a dime on the mucous surface of the lower lip. He again consulted his physician, who found, in addition to the patch on the lip, two areas on the scalp where the hair was falling out. After taking mixed specific treatment for about ten days the hair stopped falling out, and the mucous patch began to heal. There was no secondary cutaneous eruption, nor were there any further manifestations of lues.

About seven years ago, in 1908, a small bleb appeared on the lower lip, near the left angle. The patient painted it with the tincture of the chlorid of iron. The application caused very great pain, and from that time on the lesion has grown steadily, creeping slowly along the margin of the lower lip; for the past two years it has also been spreading along the margin of the upper lip. The growth then extended backward along the cheek, following and invading the old scar on the cheek. Many Wassermann complement-fixation tests have been made, and all were negative. The patient has been in the habit of taking a course of mixed treatment in the spring of every year.

COMMENTS

DR. MURPHY (June 17, 1915): This is a very interesting case, and at the same time a very unfortunate one. One must draw his conclusions as to the etiology from the physical appearance of the lesion, and the etiology controls the line of procedure. From the history one might infer that this is a specific, or spirochetic lesion on the lip, but it is *not* a spirochetic lesion. It is one of the creeping types of papilloma that begin in the mucous membrane, and it has spread over the mucosa clear up to the skin-margin, and has even spread upon the inside as well. Again, as the result of physical examination one might say that it is a carcinoma: it is *not* a carcinoma, since there is no place where it is ulcerated, nor has it ever been ulcerated. The caustic with which it was treated caused ulceration, but the lesion has never ulcerated spontaneously. It has healed up in certain areas and broken down in others, which is against its being a carcinoma. From a surgical standpoint this is a very unfortunate situation. The patient is a prominent man, and naturally does not want his face disfigured. To prevent disfigurement we must try to remove the mucosa, at the same time preserving enough for a flap to restore the vermilion border of the lip: that is where the greatest difficulty is going to be. By narrowing the angle of the mouth we hope to keep the muscularis of the lip in fairly good shape. Remember, this neoplasm has not invaded the submucous tissue: therefore it is only a papilloma. The tumor becomes carcinomatous first when its cells have penetrated the basement membrane. In the present state of development of the neoplasm, however, there is no evidence of carcinoma—none of involvement of the regional lymph-nodes. At present the Wassermann reaction is negative. There are no leukoplakic spots on the back of the throat, but on the side of the lip opposite to the lesion the leukoplakic spots that existed before the papilloma appeared are still present.

Papillomata within the mouth begin in three different places. Arranged in order of frequency, the most common site of all is the area, just behind the last molar tooth, where the mucosa falls across the erupting molar tooth and becomes repeatedly traumatized during mastication (Fig. 262). The mucosa-tabs remaining after extraction of a tooth are also subjected to the same trauma. The next common site is exactly at the angle of the mouth. The third common site is the frenum which connects the middle of the lip with the gum. On the tongue, the site most commonly involved in papilloma-formation is the frenum. The most common site of *carcinoma* of the tongue is the mucosa just behind the tip. Carcinoma, however, is an entirely different lesion from papilloma.

Now you have a good view of this papilloma: unlike a carcinoma, it is not ulcerated; not bleeding; nor have the epithelial cells penetrated the basement membrane and invaded the submucosa. Recall the patient we had a week or ten days ago with carcinoma of the buccal mucosa involving the soft palate (see previous case). He had the same type of neoplasm, as far as the *gross* appearance was concerned, but clinically the lesion was carcinomatous. If papillomata are permitted to go long enough without being treated, they will eventually become malignant. When you get a malignant neoplasm of the mucous membrane of the mouth you have the most malignant of all cancers, except those of the melanotic type. They do not respond at all to radium, nor to *x*-ray. In the Radium Institute in London they will not take a case of carcinoma of the mucous membrane of the mouth for treatment. Why? Because it has been the experience of Colonel French, who has charge of the institution, that carcinomata of that type do not respond to radium. (See footnote, p. 1029.)

OPERATION

From a pathologic standpoint, this lesion is a wart involving the mucosa, or a papilloma (Fig. 266). [Incision.] I must go through the entire thickness of the mucous membrane and skin.

. . . You see that the moment I enter the submucosa I am in perfectly normal tissue, which differs from carcinomatous in the absence of infiltration and of induration. . . . Now I am dissecting down under the mucosa on the inside, where the patch of leukoplakia is, and taking off the mucosa just the same as I took the skin off on the outside. . . . I shall excise the papilloma, and, in removing it, shall go wide of the limits of the growth into healthy tissue. I remove this papilloma in the same way as I remove a wart on the skin. If I have not taken out all the papillomatous tissue, the growth will return; and if I have not taken out all the base, the growth will return.

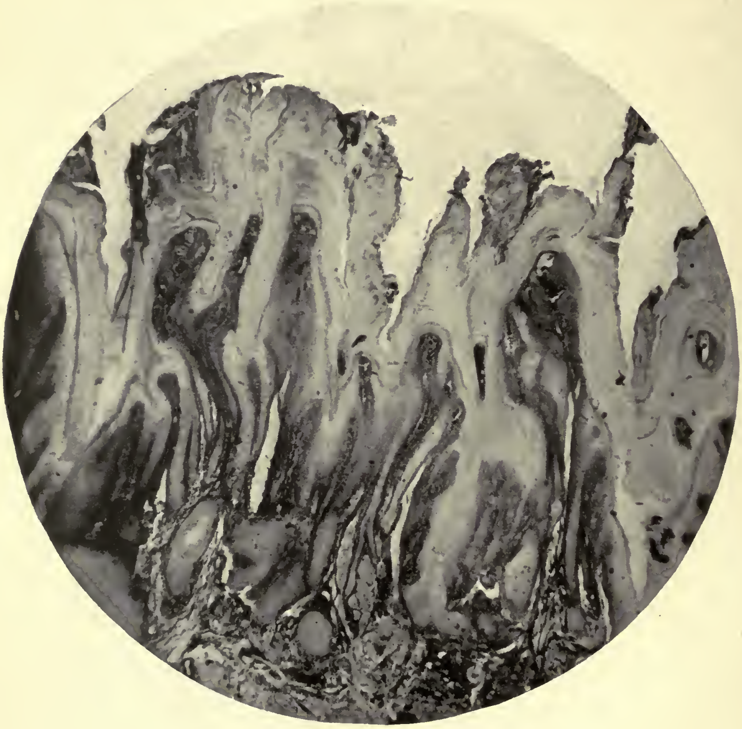

Fig. 266.—Photomicrograph of section of papilloma removed from lip. Note preponderance of fibrous tissue and papilliferous nature of tumor (×16).

Now we are ready to reconstruct the lip, and I am planning for the flap of mucosa. . . . That suture is to approximate the muscularis. You see how I am bringing up that mucosa for the vermilion border again: this flap was taken from the far angle. There is a well-shaped flap for the lower lip. See how nicely it shifts into position! Should this flap survive—and we have every reason to believe that it will—then this plastic reconstruction of the lip will be a great success. The reconstruction of the lower lip is most important. The reconstruction of the upper lip is very easily executed, because we have so much less space to close in. See what a splendid cover that flap made for his lip, and how it comes clear up!

CONGENITAL NASAL DEFORMITY—PLASTIC OPERATION—INSERTION OF BONE-GRAFT FROM TIBIA

Summary: History of previous operation; comments—condition of nose before the first operation and at present; plan of operation—scar-tissue of previous operation a handicap; importance of avoiding penetration of nasal mucosa; operation—nasal bed prepared and bone-graft from tibia inserted into it and contacted with frontal bone and rudimentary nasal bones; recapitulation; post-operative comments—importance of inserting graft *beneath* periosteum; no-hand-contacting technic diminishes chances of infection.

HISTORY OF PREVIOUS OPERATION

THE patient, a female aged eighteen years, was admitted to hospital June 14, 1915. She had a congenitally double nose, for the correction of which she was operated upon by Dr. Murphy eleven years ago—on August 25, 1904. She now returns for a second plastic operation upon the nose.

First Operation.—At the time of the first operation the skin of the nose was incised in the midline from glabella to tip, was elevated and retracted on both sides. The rudimentary nasal bones were divided in the midline with a scalpel, and the upper border of each nasal bone was separated from the frontal bone with a chisel. Both nasal bones were then elevated from their depressed position between the nasal processes of the maxillæ so as to form a bridge, and were secured by three catgut sutures which were passed through near their adjacent edges and tied. Three silkworm-gut sutures were then passed through the skin, the nasal bones, and through leaden plates on the skin, and were threaded through shot and tied down on each side. Two other silkworm-gut sutures were passed in like manner, one below through the inferior meatus and the other above through the skin and the nasal bones, shot being passed over the ends of each suture for the purpose of fixing them. The skin-edges over the nose between the silkworm-gut sutures were brought together with horsehair sutures.

Examination, made on June 14, 1915, revealed a communication in the septum leading from one side to the other.

COMMENTS

Dr. Murphy (June 16, 1915): Before the first operation, eleven years ago, the patient had a bifurcation which extended the whole distance from the frontal bone down to the tip of the nose. The nasal processes of the maxillæ stood out divergently in place of meeting in the midline over the septum. The skin over the glabella was flattened and widened. At the operation we first incised and elevated the skin and divided the nasal bones from the nasal processes of the frontal bone; then we fractured the nasal bones close to their attachment to the maxillæ, bent them over toward each other until they met in the midline, and sutured them together and the skin over them. Primary union resulted.

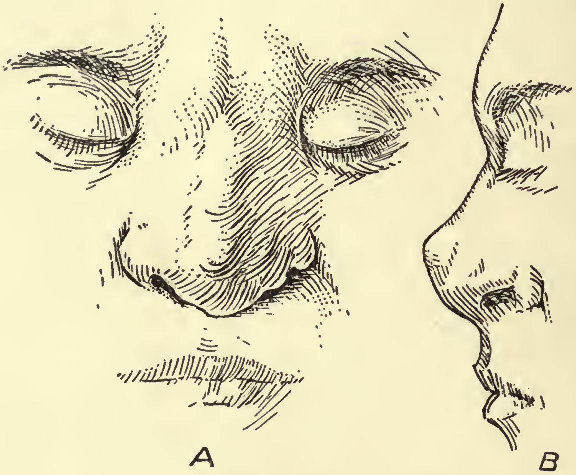

Fig. 267.—*A*, Front view, showing depression in midline with an elevation on each side at the root of the nose. Broad folds of skin overlap the inner canthus of both eyes. *B*, Profile view, showing the depression and saddle-shortening of the nose.

Now the girl returns to us because there is no bony skeleton in the nose, and because there is a folding of the skin of the forehead over the inner canthus of both eyes (Fig. 267, *A*). There is a depression in the midline at the base of the nose, with an elevation on each side. Seen from the side, the depression and saddle-shortening are evident (Fig. 267, *B*).

The operative plan to-day is to incise the tissues in the mid-subciliary ridge down to and through the periosteum on the frontal bone, free and lift the periosteum of the frontal bone and also the remnants of the periosteum of the nasal bones, and tunnel under the skin of the nose from above downward to the tip with scissors curved on the flat, and then insert a bone-transplant from the tibia between the mucosa and the skin of the nose from the frontal bone to the tip, contacting the bone-transplant with the frontal bone above, so that the transplant may be invaded by the osteogenetic process from the frontal bone, and contacting it with the rudimentary nasal bones just below the frontal (Fig. 268, *1* to *4*). Were this a normal nose, I would have no trouble in following the cleavage-plane between the skin and the mucosa, but in view of the deformity and the scar-tissue from the previous operation, I now anticipate much trouble in splitting subcutaneously accurately between these two tissue-layers. If I do not succeed in confining the dissection to the cleavage-plane without penetrating the mucosa and entering the nasal cavity, I shall have to abandon the operation and close the wound, for in that event nothing will be gained by doing anything further. Should the scissors penetrate the mucosa and enter the nasal cavity, the communication thereby established between the operative field and the bacteria-laden air within the nasal cavity will expose the bone-graft to infection, and, when infected, it will be necessary to remove the bone-graft, so that under these circumstances the bone-graft had better not be put in. Should I succeed, however, in separating the skin from the mucosa without penetrating either layer, I shall remove a bone-transplant from the tibia. On the other hand, if I should fail to separate these layers properly, then I intend performing a plastic operation upon the skin alone, and remove this redundant fold of skin from

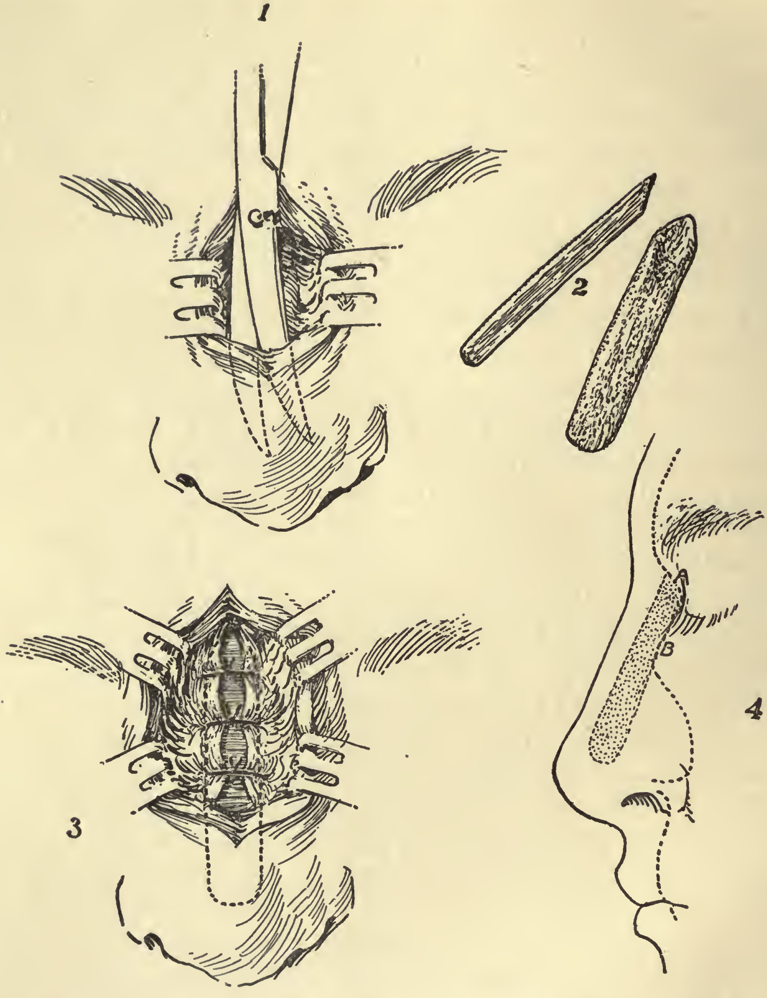

Fig. 268.—*1*, Sketch showing edges of midline incision retracted and sharp-pointed, curved-on-the-flat scissors making a dissection between the skin and mucosa of the nose. This dissection extends down to the tip of the nose, but not through the skin. It was broad enough to admit readily the bone-transplant, so that the skin and mucosa were free from tension when the periosteal and subcutaneous sutures were applied. *2*, Shows the conformation of the graft, lateral and posterior views. *3*, Shows the periosteal and subcutaneous catgut sutures in position over the graft. *4*, Diagram showing the graft inserted under the periosteum of the frontal bones and against the freshened frontal bones at *A*, and resting upon the freshened rudimentary nasal bones at *B* as a fulcrum (compare with Fig. 267, *B*). (Sketches made at operating table.)

the frontal region and the inner canthus of the eyes preparatory to inserting the bone-graft later. Considering that it was a congenital double nose previous to the first operation, it is a fairly presentable nose after the plastic operation (Fig. 267, *A*). But what the nose in its present shape needs is filling in by a bone-graft, for although it will stay up high enough as it is, yet the bony support of a nasal skeleton that will keep the tip pointing downward is lacking (Fig. 267, *B*).

Now you see what I mean by the redundant folding of the skin of the frontal region as it spreads out broadly over the inner canthus of both eyes (Fig. 267, *A*). If I accomplish nothing else, I must at least overcome the unsightliness of these frontal folds. At the time of the first operation we told the patient to return in four months to have a plastic operation performed upon these frontal folds over the canthi, and here she comes back after waiting eleven years! That is a long time to postpone matters. I would not have much trouble in splitting that cicatricial mass were it attached to the nasal septum, but, unfortunately, it is free from the septum. There is a pronounced depression of the nose, but still fairly good support is afforded by both rudimentary nasal bones, so that I have that much support as a working basis. There is no septum under this portion [indicating]. As stated above, the difficulty is to split that scar-tissue with the scissors, and at the same time keep always between the mucosa and the skin as the dissection proceeds all the way down to the tip of the nose. We performed this same operation upon a patient who did not have such an extent of scar-tissue, but who, instead, had a great depression in the nares, and obtained a beautiful result.

OPERATION

[Incision made over midsubciliary ridge ¾ inch in length and parallel with long axis of nose.] With the tip of scissors that are curved on the flat we make a slow, subcutaneous cutting dissection clear down to the tip of the nose (Fig. 268, *1*). By spreading the blades of the scissors apart as we work toward the tip a path is opened up between the skin and the mucosa ¾ inch in width all the way. This path is broader at the tip than higher up. I want to see if I have penetrated into the nasal cavity. If I have traumatized the mucosa, there will be blood in the nasal cavity. [Introduces wad of cotton on a carrier into nose and withdraws it, unstained by blood.] The cotton is dry and free of blood: that is fine. We succeeded in preparing the bed for the bone-graft without getting into the nasal cavity.

Now we shall remove the bone-graft from the tibia. Never make the incision directly over the shin, but place it to the outer side, over the tibialis anticus muscle. [Removes graft from tibia.] We leave the periosteum on the fragment we are removing so as to get the advantage of its osteogenetic influence, and we pare the graft to the proper size and shape (Fig. 268, *2*). [Adjusts bone-graft to bed prepared for it between skin and mucosa.] The nasal and frontal bones have been prepared to receive the graft—it goes into its new bed without difficulty, contacting, by its upper end, with the frontal bone, and above and behind with the rudimentary nasal bones (Fig. 268, *4*). You see I am thrusting all the tension upon the connective tissue and none upon the skin or mucosa. We now suture the periosteum and subcutaneous tissue over the graft (Fig. 268, *3*), so that its upper end is practically entirely surrounded by osteogenetic influences. We close the skin by horsehair sutures.

I would appose the skin-edges with Michel clips were it not for the fact that clips occasionally set up irritation. If there is the slightest excess of scar from such an irritation, these young women would become very sensitive about it. If the field remains sterile, and if the bone-graft does not ulcerate through the periosteum and skin, then the patient should have a good result. The soft part of the nose is the most favorable tissue in the body in which to bury a transplant, and should that transplant live through osteogenetic influence from above, it will be a source of very great satisfaction (Fig. 269).

RECAPITULATION

Let the record show that the incision was made in the line of the old scar down to the periosteum and including it; that the periosteum was then split clear down to the tip and elevated from the nasal bones, which had united eleven years ago, and when that step had been completed, we divided or split the tissues in a plane between the skin and the mucosa without getting into the cavity of the nose; that we obtained a suitably shaped piece of bone from the tibia and succeeded in implanting it into its prepared bed between the skin and the mucosa without opening

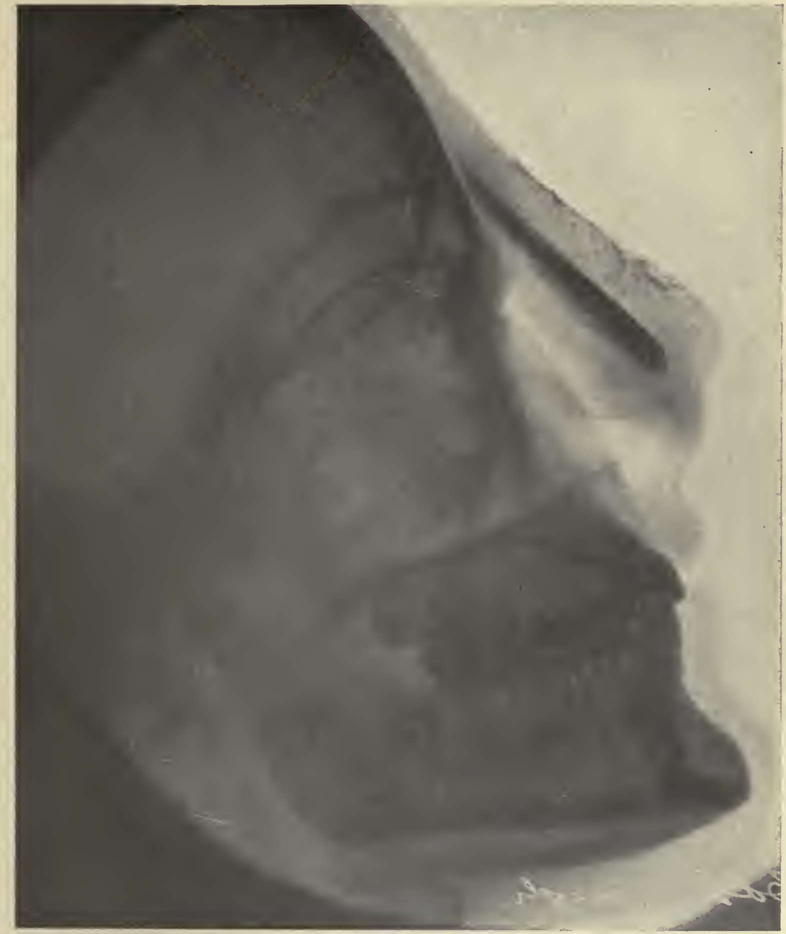

Fig. 269.—Postoperative skiagram showing bone-graft *in situ*. (Profile view.)

into the cavity of the nose (Fig. 268, *4*); and finally that we believe we succeeded in performing the operation without hand-contacting, thereby diminishing the chances of infection.

POSTOPERATIVE COMMENTS

I would not expect that bone-graft to regenerate unless I had inserted it beneath the periosteum. After the bone-graft has become healed in I can just split the superficial tissues down subcutaneously, take some deep fascia and fat from the thigh or abdomen, and interpose it between the mucosa and the skin, thereby filling out the soft parts of the nose as much as I please. We succeeded in removing the redundant fold of skin from the inner canthus of both eyes.

[A plastic operation upon the skeleton of the nose was published in the Clinics for August, 1915, pp. 593–597; a plastic operation upon the soft parts of the nose appeared in the same number, pp. 589–591.]

CARCINOMA OF MAXILLARY ANTRUM—EXCISION OF MAXILLA

Summary: Clinical history of patient; examination; comments—chronicity of lesion; danger of traumatizing a neoplasm; first operation—preliminary ligation of external carotid artery; excision of maxilla by Fergusson's method; recapitulation; postoperative comments—importance of early microscopic examination of neoplasm; sarcoma of maxilla offers favorable prognosis if removed early; second operation—plastic upon soft palate; postoperative comments—danger of clots forming in pharynx; advantages of ether-olive-oil anesthesia when operating upon nose or mouth.

CLINICAL HISTORY OF PATIENT

THE patient, a male aged fifty-two years, was admitted to hospital April 28, 1915. The family history revealed that two sisters and one brother died of pulmonary tuberculosis. The past history is negative.

Twelve years ago, in 1903, the patient noticed an enlargement of the gum, the size of a small pea, just above the right upper bicuspid tooth: no ulcer was present. During the next eight years the swelling increased very slowly in size. About four years ago, on July 11, 1911, the gum began to pain, and at the same time a physician excised the growth on the gum. The traumatic ulcer left remaining after the excision healed over in about two weeks. Two months later, in September, 1911, the right maxillary antrum became painful. There were no chills, nor was there any fever or swelling, but the antrum remained sore and tender. Two months after the antral signs began a physician, operating, entered the antrum from the buccal cavity just above the bicuspid tooth, and inserted a gauze drain. There was but slight discharge, however, and the antrum continued to ache. For the next two years the pain continued, and the patient treated the involved area himself by the use of mouth-washes and hot applications to the cheek.

Seven months ago, in September, 1914, a physician opened the antrum by approaching from the outer (cutaneous) surface of the cheek, and curetted its bony walls. Again there was but slight discharge after operation, and the cheek and border of the gum continued aching. In the middle of February, 1915, the antrum was reopened through the old cutaneous incision, curetted, and the opening into the mouth above the bicuspid tooth was enlarged. The aching in the cheek and in the maxilla continued, however. At the present time there is a scanty purulent discharge from the sinus.

Examination.—A specimen removed by Dr. Murphy was microscoped and reported to be an adenoma, and one that was strongly suggestive of malignancy. Both Wassermann and luetin tests were made and were negative.

COMMENTS

Dr. Murphy (April 30, 1915): That history indicates a very chronic condition, and, from the clinical course, shows that the correct diagnosis had never been established. It is now twelve years since the beginning of the antral disease. The patient was fairly comfortable until the futile and meddlesome operations, by their trauma, stimulated the neoplasm to unbridled and riotous growth.

Illustrative of the danger of traumatizing a neoplasm is the behavior of a melanotic tumor when subjected to trauma. A melanotic tumor, whether in the mouth, on the back, or on the cheek, must be removed *in toto* without being traumatized. To avoid traumatizing the tumor you must go wide of it, and you must get it all out. If you do not, you may be sure of a fatal termination. We have had scores of similar cases. For years their course was apparently benign, until they were meddled with, and the moment they were meddled with or traumatized, *that* moment the fire started.

This tumor extends almost to the midline of the palate, and has been of very slow growth. The patient is suffering greatly and is incapacitated by the lesion.

FIRST OPERATION

[Oblique incision made on right side along anterior border of sternomastoid below angle of mandible, as for ligation of external carotid artery.] The lymph-nodes here are enlarged sufficiently to arouse suspicions of malignancy. . . . There is the facial vein. This preliminary incision is for the purpose of ligating the external carotid artery, in order to diminish the amount of hemorrhage during the remainder of the operation. . . . There is a landmark—the digastric muscle—shining in the field. . . . Now we are opposite the greater cornu of the hyoid bone, just to the inner side of the external carotid artery. By means of a spreading dissection we isolate the external carotid artery and ligate it.

[Fergusson's incision, made from inner canthus down along nasofacial and then nasolabial grooves to and through philtrum of lip; next from inner canthus horizontally along lower orbital margin to end over malar bone beyond outer canthus.] I am making this incision follow, in part of its course, the old scar-line. . . . I am now close to the infra-orbital nerve, in the territory of which the patient has had most of the pain: this nerve supplies the whole maxilla and the lower eyelid, cheek, and lip anterior to it with sensation. [Dissects up the flap and retracts it outward, elevating soft tissues from maxilla subperiosteally.] I have now exposed the maxilla: note how slight the hemorrhage is. [Using a Gigli saw, divides the nasal, the orbital, the malar, and the palatal processes of the maxilla, and with the lion-jawed forceps wrenches the maxilla with its contained tumor free from the pterygoid process of the sphenoid bone and the tuberosity of the palate bone.] Before closing the wound I shall apply the cautery lightly over the field. You see that, as we replace the flap and suture it, the amount of deformity is very slight. [Wound closed with horsehair sutures, and dry dressing applied.]

RECAPITULATION

Let the record show that the growth involved the maxillary antrum, and that, notwithstanding the microscopist's report of benign adenoma, yet to the naked eye the growth resembles carcinoma more than adenoma; that after making the usual incision along the nares through the old scar-line, we freed the upper flap and retracted it outward; that there was not much bleeding, since the external carotid artery had been ligated; that the maxilla was exposed fully; that the blood was kept out of the pharynx until I was ready to wrench the maxilla free with the heavy forceps; that this step was easily accomplished after separating the nasal, orbital, malar, and palatal processes with the Gigli saw and bone-cutting forceps, and finally that the maxilla was avulsed together with the tumor.

POSTOPERATIVE COMMENTS

This is a late period in the course of the growth to be performing this operation: operative measures should have been considered at the time of the very earliest manifestations of the tumor—at least a specimen should have been removed and microscoped before the first operation of incision and drainage. Excision of the maxilla is not nearly so disfiguring to the patient as is excision of the mandible.

The most common tumor of the maxilla—and the one that offers the most favorable opportunity for complete removal by an early, radical operation—is sarcoma. Many years ago I excised a maxilla for sarcoma, and it is surprising how little deformity the patient presents now: one could scarcely tell that the maxilla was absent. Excision of both maxillæ, however, gives rise to marked deformity.

SECOND OPERATION, May 19, 1915

This patient was operated upon three weeks ago. After the previous operation, upon inspecting the field, we concluded that we had preserved a little too much of the soft palate. At the present sitting, therefore, I wish to perform some plastic work upon the soft palate and remove a spiculum of bone that projects into the pharynx. We removed the entire hard palate on the right side.

[Pares redundant palate.] I am endeavoring to prevent clot-formation in the pharynx. We had one experience with a clot being inhaled and lodging at the bifurcation of the trachea, and that experience was sufficient to last us a lifetime, notwithstanding we saved a life. Now I have this field prepared and shall proceed to close it. . . . I think I can show you what we have done. We have enlarged the opening and resected a larger amount of the soft palate; we then swung the remainder of the soft palate over and fastened it to the inner side of the cheek. When the process of healing is completed, the dentist will fit a rubber obturator in the palatal defect so that there will be no further leakage from the nose into the mouth.

For this operation we employed the ether-olive-oil method of anesthesia, which is most convenient when working about the nose or mouth.

[*N. B.*—The patient did not come out of the anesthetic as promptly as was expected, so that, as a precautionary measure, tracheotomy was performed. Later on he emerged from the anesthetic without any untoward symptoms, and convalescence was uneventful.]

[A case of inoperable sarcoma of the maxilla, treated by salvarsan and sodium cacodylate, was published, with illustrations, in the CLINICS for April, 1912, pp. 208–209; a case of inoperable recurrent carcinoma of nasopharynx involving both maxillæ, ethmoid, frontal and malar bones, successfully treated with mixed toxins by Dr. Coley, was published, with illustrations, in the CLINICS for October, 1915, pp. 957–967; a case of sarcoma of the malar bone with illustrations was published in the CLINICS for June, 1915, pp. 549–566; and a case of osteomyelitis of the maxillary antrum appeared in the CLINICS for August, 1915, pp. 613–614.]

CONGENITAL (THYROGLOSSAL DUCT) SINUS OF NECK—TOTAL EXCISION OF SINUS

Summary: Clinical history of patient; comments—discussion of differential diagnosis with intern; embryologic explanation and course of a thyroglossal duct sinus; sublingual thyroid gland; operation—necessity of total excision of a congenital epithelial-lined sinus, and futility of any other method of treatment; table of tumors of neck.

CLINICAL HISTORY OF PATIENT

THE patient, a male aged nineteen years, was admitted to hospital April 29, 1915. The family and past histories are negative.

Ten years ago, in 1905, for the first time the patient noticed a small lump the size of a hazel-nut a little below the thyroid cartilage, and to the left of it: neither chills nor fever were present. The swelling disappeared and later reappeared—this cycle occurring about every four weeks for one year.

A year after the patient first noticed the swelling a physician, operating, lanced the swelling, and the wound discharged pus for one week and then healed. Immediately after the operation a papular skin eruption appeared on the face and scalp and remained for ten days. Three months after the operation the scar opened itself and began discharging, the discharge continuing for a period of eight years—up until one week ago, when it closed and remained closed for three days. With the closure of the wound the front portion of the neck became tender to pressure. At the end of three days the wound reopened and then closed for two days.

The general health has always been good, and the patient has lost no weight. There are neither symptoms nor signs of thyrotoxicosis.

COMMENTS

Dr. Murphy (May 3, 1915): How long did the patient have that scar on his neck?

Intern: He said that it had been there for two years.

Dr. Murphy: Did he have the scar on his neck before he was operated upon?

Intern: Dr. Hochrein, who wrote the office history, says that there was no scar there until after the operation.

Dr. Murphy: What is the matter with him?

Intern: I think that he might have a congenital lesion, but the fact that he did not notice it until he was ten years old speaks against its being one. There might be an abscess in the isthmus of the thyroid gland.

Dr. Murphy: Had there been an abscess in the thyroid gland, he would have had chills, fever, and other symptoms of toxic absorption from the infection. I think that it is tuberculosis. Show me the error of my ways: why would tuberculosis occur in the median line, or why would it not occur there?

Intern: On account of the distribution of the lymph-nodes in the neck I should think that tuberculosis would not occur in the median line.

Dr. Murphy: Why not?

Intern: Because tuberculosis would have to come from a breaking down of the lymph-nodes or from extension from a near-by focus of tuberculosis.

Dr. Murphy: What do you mean by extension from a near-by focus?

Intern: The lymph-node nearest the portal of infection becomes infected, and the infection extends to the other lymph-nodes.

Dr. Murphy: All right. Why could it not be that?

Intern: Because there are no lymph-nodes located in that zone

Dr. Murphy: Correct. What else?

Intern: He might have had a pyogenic infection with an abscess forming in the isthmus of the thyroid gland.

Dr. Murphy: Yes; he might have had a pyogenic infection of the isthmus of the thyroid gland. If it were an abscess in the isthmus, why should the infection have continued up to the present time?

Intern: I do not think that it would have.

Dr. Murphy: Why not?

Intern: Because he would have had more marked symptoms.

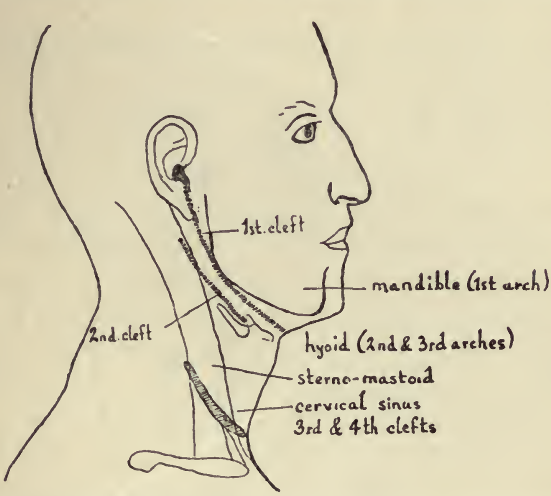

Fig. 270.—Diagram showing orientation of branchial clefts to structures in a fully developed neck. "If the development of vessels be normal, a fistula of the second cleft must lie between the external and internal carotids; third cleft, common carotid, and vagus; fourth cleft, bend around the subclavian on right side, arch of aorta on left side. The last-named is very unlikely to arise, and, indeed, it is only fistulæ of the second cleft that have hitherto been recognized with perfect certainty" (McKenty).

Dr. Murphy: A simple pyogenic infection does not run such a chronic course, but soon heals of itself. If, however, a sinus communicates with a calcareous tuberculous lymph-node, the calcareous material prevents absorption, and if healing finally does take place, the sinus may recur below that point and appear lower down. Recall the fact that in the patient from Iron Mountain, Michigan, last year, the discharge of pus continued until we took out the foreign body. What do you now think the lesion may be?

INTERN: I think that it may be a congenital lesion.

DR. MURPHY: What do you mean?

INTERN: In the median line and adjacent area of the neck a congenital lesion is most commonly due to failure of certain embryonic clefts to close.

DR. MURPHY: You have the branchial clefts coming from the side toward the midline (Fig. 270). Then you have what else?

INTERN: You have, in addition, the thyroglossal duct.

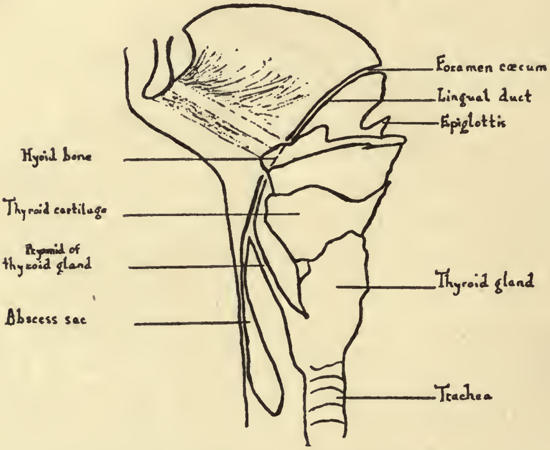

Fig. 271.—Diagram of abscess of persisting thyroglossal duct, which runs posterior to or occasionally through the hyoid bone, explaining why it is so difficult to remove the duct surgically in its entirety (McKenty).

DR. MURPHY: The thyroid gland is formed in the embryo at the base of the tongue, and between the two lobes of the tongue, at the site of the *foramen cæcum:* it soon migrates lower down in the neck—even down into the chest. As it descends it occasionally trails the epithelial lining all the way from the base of the tongue to its point of arrest, and you will see a thyroglossal sinus remaining in the track of the descended thyroid gland, and this epithelial-lined sinus may run clear up to the foramen cæcum of the tongue (Fig. 271). Occasionally you will find the sinus communicating at one or two places with the surface of the body, or with the cavity of the trachea, or with the cavity of the larynx between the hyoid bone and the thyroid cartilage. At other times the sinus will lead from its termination below up to the body of the hyoid bone. Not infrequently you will find that the thyroid gland does not migrate downward at all, but remains under the tongue throughout life, forming a sublingual thyroid gland. If this sinus is congenital, has it not been here since birth?

INTERN: I think that it must have been.

DR. MURPHY: Why has the patient not known of its presence before? Why did he not notice it until he was nine years old?

INTERN: The orifice of the sinus was there, but it was not discharging.

DR. MURPHY: That is correct. The epithelial cells lining the sinus were there. This scar resembles a congenital scar, such as one sees around the bladder in cases of exstrophy of the bladder. The right lobe of the thyroid gland is of fair size, but the left lobe is not so large. In performing an operation for a congenital sinus I know where I am going to begin the dissection, but I do not know where I am going to end it. One must follow the sinus to its ultimate termination. What is the nature of the cell-lining of a congenital neck-sinus? It is epithelial, and therefore will never heal spontaneously. Were the sinus the sequence of an infection, and therefore lined with connective-tissue cells, then it *would* heal spontaneously. Here is the pocket of the sinus, and it is of the same character as that of the pilonidal cysts, which occur in the postanal region, and which, also congenital in origin, are lined with epithelial cells and are never cured except by an accurate and complete dissection, with total removal of the sinus.

When the thyroid gland does not descend, but remains up under the tongue, it is subject to the same pathologic changes that it would be were it in the usual situation, or even if it had descended into the chest. In one of these cases of sublingual thyroid the patient came to me presenting a tumor beneath the middle of the tongue. I did not suspect that the tumor was the thyroid gland, but diagnosed a tumor of the thyroglossal duct.

Operating, I ablated the "tumor," which, on examination, was about as large as an English walnut and lobulated. I recognized macroscopically on sectioning it that it was a thyroid gland, and that it was undergoing calcareous degeneration at one point, while presenting the typical colloid accumulation at another. After the operation, when we began to search for a thyroid gland in the usual situation, we found, to our dismay, that the patient did not have one there—we had removed the only thyroid gland she had! We kept her in the hospital, and, sure enough, in the regular time for signs of hypothyroidism to appear—between four and one-half and five weeks—first the lips and next the skin of the hands began to get a little thicker, and then she developed typical myxedema, with loss of axillary, suprapubic, and eyebrow hair. The only thyroid tissue she had was the undescended glandular structure under the tongue, and that we removed by operation. We carried her through on thyroid treatment for about fourteen months, until enough thyroid cells had proliferated from some fragments we had evidently left beneath her tongue to overcome the hypothyroidism and reëstablish the metabolic balance of the ductless glands. We had recognized the situation at once, and instituted the proper measures—otherwise she would have died of myxedema.

OPERATION

[Incision around orifice of sinus, and dissection along path of sinus.] . . . Now we have taken out all that epithelial-lined track that we could recognize. If it is not wholly removed, it will again begin to discharge, just as before operation. No amount of cauterization nor of curettage will heal it—nothing short of complete removal. Recognizing its congenital and epithelial-lined nature, one has no difficulty in outlining what should be done; but one often has great difficulty in removing the sinus entirely. Now we shall mop this field with 5 per cent. phenol. After excision of this sinus we shall have a considerable amount of tension upon the skin-edges, and this tension will be present for a long time after operation. I excised the original scar, and successfully to contact the skin-edges over the resulting gap I have to put considerable tension upon the skin. In order to avoid such an unsightly scar as originally existed in his neck I insert the needle into the skin far back from the skin-edges, using a double strand of horsehair suture. The wound will be dressed with collodion alone.

[In the Clinics for April, 1914, pp. 285–302, there was published a talk "On the Embryology, Anatomy, and Physiology of the Thyroid Gland."]

TUMORS OF THE NECK

A. Congenital:

1. Branchiogenic cysts or sinuses; supernumerary auricle.*
2. Thyroglossal cysts or sinuses: aberrant thyroid glands.
3. Lymphatic cysts: lymphangiomata.
4. Grumous or hemorrhagic cysts: hemangiomata.
5. Bursal cysts: hygromata.
6. Teratomata.

B. Infections:

1. Furuncle and carbuncle.
2. Ligneous phlegmon of Reclus.
3. Secondary lymphadenitis (primary focus in nose; pharynx; tonsils; tongue; teeth; lips; face).
4. Secondary suppurative lymphadenitis:
    - (*a*) With cellulitis and phlegmon—Ludwig's angina.
    - (*b*) Submental and submandibular.
    - (*c*) Submastoid and paratracheal—Dupuytren's phlegmon; cervical and substernal cellulitis.
    - (*d*) Para-esophageal.
    - (*e*) Retropharyngeal (infantile cervical Pott's disease).
5. Tuberculous lymphadenitis.
    - (*a*) Simple tuberculous infection.
    - (*b*) Mixed tuberculous infection.
    - (*c*) Tuberculous ulcer.
    - (*d*) Tuberculous sinuses of
        1. Lymph-node origin.
        2. Osseous origin.
6. Spirochetic lymphadenitis.
7. Actinomycosis; blastomycosis; mycotic abscesses (mycelia).

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* Of 30 congenital tumors of neck McKenty (*Surg., Gynec. and Obstet.*, Aug., 1914, 145) found the following varieties:

A. Branchiogenic cysts	15
B. Branchiogenic carcinoma	5
C. Thyroglossal cysts	9
D. Carotid body tumor	1
Total	30

8. Echinococcus cysts.
9. Anthrax.

C. Non-malignant Tumors:
1. Sebaceous cyst; lipoma; fibroma; chondroma; osteoma.
2. Aberrant thyroid gland; physiologic hypertrophy of thyroid; goiter.
3. Aneurysm of aorta and of carotid arteries.
4. Tumors of larynx.

D. Malignant Diseases:
1. Malignant lymphoma; pseudoleukemia.
2. Lymphosarcoma.
3. Carcinoma; branchiogenic carcinoma.
4. Malignant perithelioma of carotid body—operative mortality of 25 per cent.

E. Tumors of the Salivary Glands:
1. Parotid.
    (*a*) Chondroma; fibroma; endothelioma; mixed tumors ("first or second arch tumors," McKenty); Mikulicz's disease.
    (*b*) Sarcoma—fibro-, myxo-, chondrosarcoma.
    (*c*) Carcinoma of—(1) gland; (2) duct.
2. Infectious—epidemic parotiditis; interstitial parotiditis.
3. Sublingual and submaxillary—Mikulicz's disease; carcinoma; sarcoma.
4. Retention cysts—ranula, etc.

BILATERAL CERVICAL RIBS—EXCISION OF RIB ON RIGHT SIDE

Summary: Clinical history of patient; examination; comments—cervical ribs usually first cause symptoms in patients beyond thirty years of age; symptoms and signs of cervical ribs in order of appearance; subclavian artery, elevated into a superficial position by the cervical rib, simulates an aneurysm; operation; postoperative comments—topographic anatomy of cervical rib; brachial plexus and subclavian artery compressed between tip of cervical rib and scalenus anticus muscle, vein escaping: therefore, should gangrene arise, it is always dry.

CLINICAL HISTORY OF PATIENT

THE patient, a female aged fifty-four years, was admitted to hospital April 27, 1915. The family and past histories are negative.

Five years ago, in 1910, the patient noticed for the first time an unusual filling out of the right supraclavicular fossa, and at times felt a thumping in this area. A physician advised her to ignore the lesion unless it should become more troublesome.

About one year ago, in March, 1914, the right arm began to ache periodically,—every three to five days,—and there was the sensation of needles sticking into the extensor surface of the right hand. The swelling in the right supraclavicular fossa became more prominent, and the patient felt a throbbing sensation which has continued ever since.

For the past three months, since January, 1915, the aching in the arm has become more constant. The right hand feels cold at times. The second and third fingers of the right hand ache and are sensitive to pressure. The arm has never been swollen, although the patient states that at times it feels full and stiff. With the exception of having headaches periodically,—every six weeks, and of two days' duration,—she feels quite well.

Examination by Dr. Murphy (January 8, 1915).—Beneath the right subclavian artery there is a large, bulging mass which presses so closely against the posterior border of the clavicle that one could not insert the finger between the two. The subclavian artery is carried from ¾ to 1 inch above the clavicle, and arches suddenly over a hard, bony prominence. A skiagram (Fig. 272) taken at this time showed, on the right side, a long seventh cervical rib emerging over against the clavicle; and on the left side a shorter seventh cervical rib, going down vertically at a right angle. The arch of the aorta was shown to be slightly displaced, but there was no evidence of the presence of an aneurysm.

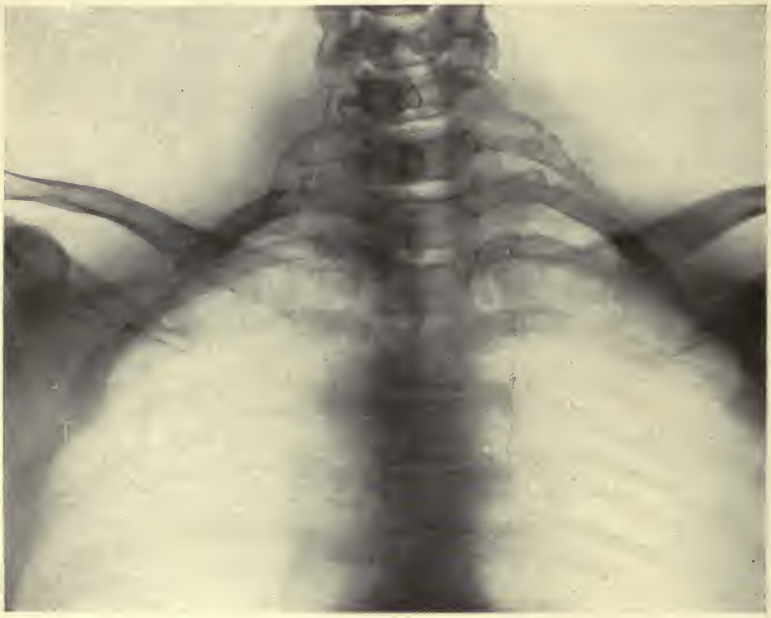

Fig. 272.—Preoperative skiagram showing bilateral seventh cervical ribs. The rib on the left side is shorter, and passes down vertically at a right angle (posteroanterior view).

At the time of admission the subclavian artery was found spread out until it was ¾ inch in diameter; by its pulsations it simulated an aneurysm. It was situated behind the tip of the seventh cervical rib.

COMMENTS

Dr. Murphy (April 29, 1915): What is the matter with the patient?

Intern: There is a long cervical rib on the right side, giving rise to pressure upon the neurovascular bundle.

Dr. Murphy: Yes. The history is classic. She is a woman of fifty-four years; but while cervical ribs are congenital, yet in the majority of cases reported in the literature the patients who came for treatment have been beyond thirty years of age. They complain of about the same symptoms: first, of the periodic sensations of numbness, appearing usually in the morning; secondly, of sensations of coldness, more marked when the hand has been continually used for a long time; thirdly, of shooting pains down the arm; fourthly, of the effects of ischemia, which at times is confined to a portion of the hand, and at other times involves the whole hand; and, finally, of the effects of atrophy and then of gangrene of one or more fingers, the gangrene eventually extending up the hand toward the elbow. Most of the sufferers complain of nerve symptoms, and most are treated for essential neuritis.

A very interesting example of cervical rib was found in a patient who came into my office two weeks ago to-day. The patient suffered first from neuritis, and had consulted a nerve specialist. The neurologist began treatment with injections of a 12 per cent. solution of phenol in alcohol and water into the base of the neck, endeavoring to reach the brachial plexus. The injections alleviated the pains in the shoulder and on the inner side of the arm, but the pains which extended down into the fingers continued. The neurologist, again injecting, endeavored to block the median nerve. I do not know what he struck with the needle, but immediately after the needle was pushed into the tissues a sharp, intense pain began. The hand fell limp at the side, and the pulse became feeble. The arm began to swell and continued swelling rapidly until it reached an enormous size. Eventually the circulation was in part restored and the swelling subsided, but there appeared gangrene of all the fingers. The patient was a prominent lady from New York city. I tried to straighten the situation out and rectify matters as much as I could. The question arose whether or not the original pain was due to a cervical rib, and whether or not the gangrene of the fingers resulted from the injection. But the gangrene did not appear immediately—not until three weeks after the injection had been made. If it were phenol necrosis from the 12 per cent. phenol solution, it seems to me that the gangrene should have appeared within a shorter time after the injection. From the history it looked to me as though the contents of the syringe went into the artery, because the arm began to swell suddenly, but soon the swelling disappeared entirely. Was there an obliterating endarteritis which involved all these large branches of the subclavian artery, from the 12 per cent. phenol injections, or were the circulatory disturbances the result of pressure from the cervical rib?

Upon examining this patient the subclavian artery is found spread out over the base of the supernumerary rib, and by its superficial position and pulsations it feels like an aneurysm. I remember that one of my early cases of this nature, when the pathologic entity was first recognized, was strongly suggestive of an aneurysm by reason of this superficial position of the artery. Cabot has said: "A pulsating mass above the clavicle means cervical rib nine times out of ten."

OPERATION

Let the record show that, after making an incision over and parallel with the anterior border of the scalenus medius muscle, we freed the scalenus anticus high up on a level with the upper margin of the seventh cervical rib, and then, by blunt dissection, reached a position behind the brachial plexus, encountering and ligating the transverse cervical artery and vein; that we then came upon the neck of the cervical rib, and with the periosteotome freed it outward toward its tip, retracting the vessels and nerve-trunks inward, but allowing the periosteum to remain on the rib; that next we freed a portion of the muscle that was attached to the rib, divided the rib at its neck with a short, bone-cutting forceps, grasped the shaft of the rib with sequestrum forceps, and avulsed the rib; and finally that the wound was closed in the usual way. The cartilaginous tip which remains attached to the first rib after the avulsion can easily be snipped off with rat-toothed Rongeur forceps.

POSTOPERATIVE COMMENTS

The patient has a cervical rib on the left side also, but by the skiagram you see that it is a very short one.

In the topographic anatomy of cervical rib the scalenus anticus muscle plays the dominant rôle in the train of symptoms and signs because the cervical rib, growing from behind forward, encounters the scalenus anticus and compresses against it, in turn, first, the brachial plexus, causing the neuralgia of which these patients complain; and, secondly, the subclavian artery, causing disturbances of the radial pulse, ischemia, and even *dry* gangrene. The subclavian vein is situated anterior to the scalenus anticus muscle, and so it cannot be caught and compressed by the rib in its growth; therefore, cervical rib never causes moist gangrene, but always *dry* gangrene, because it is not the venous circulation that is involved, but the *arterial.* The subclavian artery is stretched out over the rib to such an extent that if you put traction upon the arm you can immediately shut off the pulse by completely obliterating the lumen of the vessel at the site where the rib is producing the pressure.

[A case entitled "Double Cervical Rib" was published in the CLINICS for June, 1912, pp. 389–393.]

OSTEOSARCOMA OF SCAPULA—TOTAL EXCISION OF SCAPULA

Summary: Clinical history of patient; comments—examination of skiagram and of patient; plan of operation—excision of scapula preserving ligaments to support arm; citation of a previous case of sarcoma of scapula with tardy metastasis to spine; operation—exposure of structures about shoulder; autopsy statistics of sarcoma of femur; percentage of cases of cancer without demonstrable metastases at autopsy; further dissection; closure of wound; recapitulation; postoperative treatment. Additional note on a similar case. Table showing site-incidence of bone sarcomata.

CLINICAL HISTORY OF PATIENT

The patient, a female aged twenty-seven years, was admitted to hospital January 14, 1915. The family, past, and menstrual histories are negative. The latest mensis occurred on October 3, 1914, three months ago.

The present disease began seven years ago, in the winter of 1908. While playing basket-ball, the patient reached upward, at the same time feeling a snap in the left shoulder, followed by pain. She continued in the game, however. There was no edema or swelling of the arm, but the pain, which was dull and aching, continued two weeks. For one year after the injury she was unable to lie on the affected shoulder.

After one year the pain and discomfort subsided entirely, and she was free of symptoms until four years later, in the spring of 1913, when the left shoulder began to ache, becoming worse the following winter. In June, 1914, she had osteopathic treatments without relief. In the fall of 1914 the pain became dull and radiated to the neck, shoulder, and down the arm. The pain caused restriction of the hitherto free movements of the left arm.

Since September, 1914—four months ago—she has been unable to use this arm. She consulted a physician, who declared, upon the basis of a skiagram, that there was luxation of the left humerus.

COMMENTS

Dr. Murphy (January 16, 1915): When you look at this skiagram you note, first, that the shoulder is low down; secondly, that there is an excavation in the body of the scapula resembling a cyst or the cavity of an aneurysm; thirdly, that there is a pathologic fracture of the spine of the scapula (Fig. 273). When inspecting the patient, the whole shoulder seems to have dropped (Fig. 274). There is a tumor here that shows beautifully (Fig. 275). According to the history, she was injured seven years ago: at the site of that old injury a malignant lesion, which involves

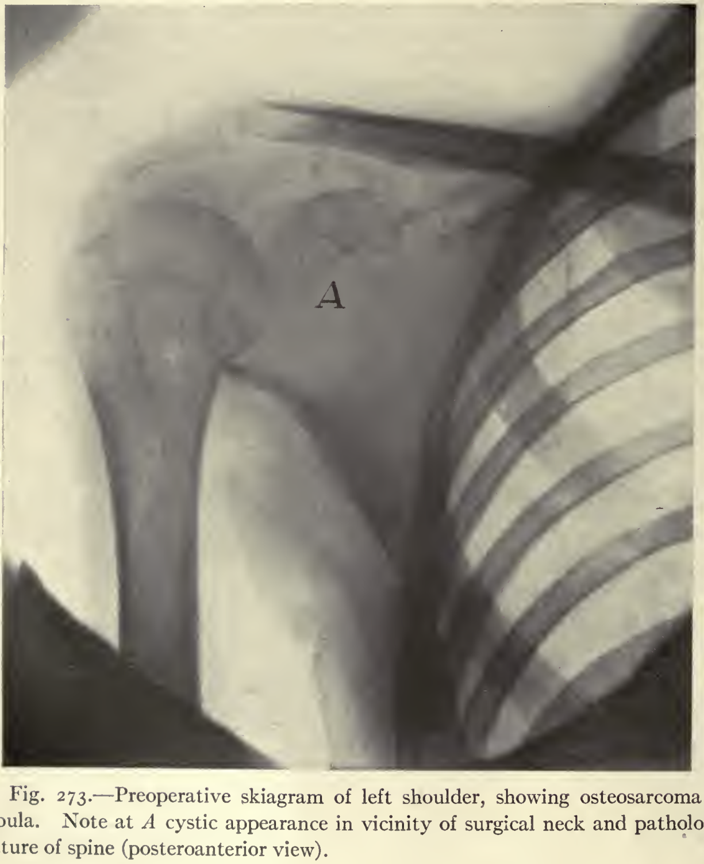

Fig. 273.—Preoperative skiagram of left shoulder, showing osteosarcoma of scapula. Note at *A* cystic appearance in vicinity of surgical neck and pathologic fracture of spine (posteroanterior view).

the scapula, has sprung up. The glenoid cavity appears intact, and although the neoplasm does not involve the acromion or the coracoid process, still we shall remove both. We shall divide the two muscles attached to the coracoid and displace them with the artery and nerves forward, excising the entire scapula, including the coracoid process. The extent of the operation depends, however, altogether upon the appearance of the bone after exposure. It is easily seen that, by preserving the coracoacromial, the coracohumeral, and the coracoclavicular ligaments with the humeral portion of the capsule of the joint and reattaching them to the clavicle, much support will be afforded the arm.

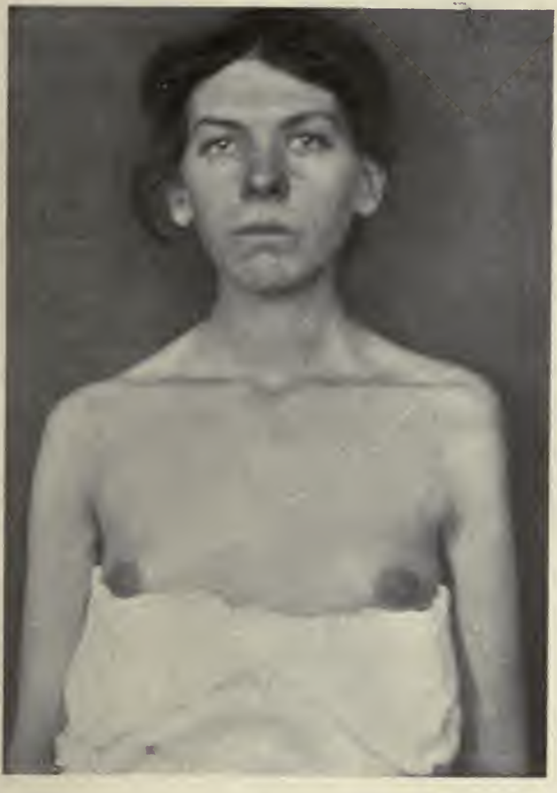

Fig. 274.—Osteosarcoma of left scapula before operation. Note dropping of shoulder. Anterior view gives no index to extent of deformity.

Seven years ago, on August 29, 1908, we operated upon a woman with sarcoma of the spine of the scapula, and excised the entire scapula, retaining the humerus. The patient could not abduct the arm from the side, but had control of other movements of the arm. After the operation she progressed satisfactorily for two years and four months, when she came back, affording me the opportunity of demonstrating her condition one Saturday morning, before the members of the Chicago Surgical Society. That morning I referred to her case an an example of cure of sarcoma of the scapula by total excision. On Wednesday, four days later, she suddenly developed complete paraplegia. One

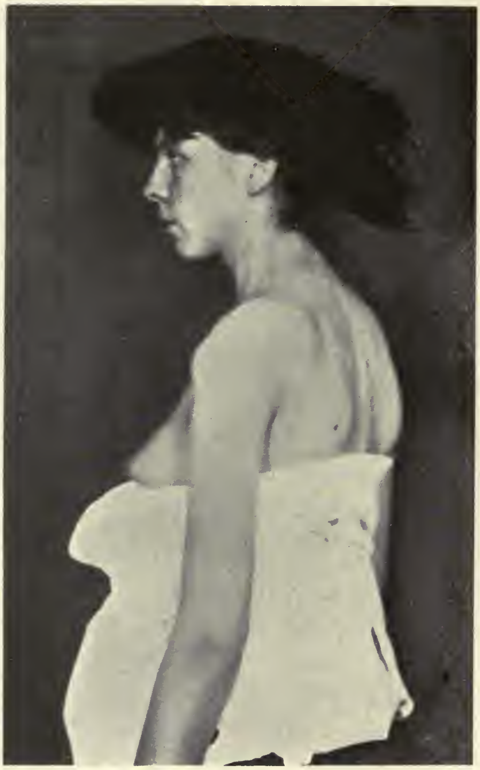

Fig. 275.—Osteosarcoma of left scapula before operation. Profile view, showing extent of deformity, most marked opposite spine of scapula.

might have suspected hemorrhage into the cord as the basis of the paraplegia, but that is the natural course of these spinal tumors. For a time they present no symptoms when within the cord, but suddenly the patient is paralyzed. Now, unless one were familiar with the course of these lesions, he would strongly suspect hemorrhage. It is not hemorrhage, however. Three days after the appearance of the paraplegia, on November 18, 1910, we operated upon her and removed a sarcoma from the spinal cord. Nevertheless the malignant process was unchecked, and she died. While I cited the case as a two year and four months' cure, yet within four days she was paralyzed! That was a shock both to the physician and to the patient.

OPERATION

These operations are tedious and necessarily painstaking. We shall estimate the size of this tumor. The head of the humerus is luxated anteriorly into the axilla, and is lodged against the neurovascular bundle. The clavicle is not involved. We shall attack from behind forward, using Kocher's approach to the shoulder-joint, although this is not the method we usually employ. Here is the posterior margin of the deltoid muscle: we must remove most of it, owing to its penetration by the sarcoma. We also free the trapezius muscle at its attachment to the spine of the scapula. There are the clavicular fibers of the trapezius: here is the serratus magnus muscle. Now we enter the shoulder-joint. That is the acromion process: this is the supraspinatus muscle, separation of which exposes the suprascapular artery. There is the long head of the biceps flexor cubiti. Next we come to the subscapularis muscle, which must be removed because attached to the scapula. Now we excise the levator scapulæ. Here is the long head of the biceps, which has just been freed. The coracoid process is now enucleated, retaining the periosteum on the bone, so that the process is readily detached. The bleeding has practically entirely stopped. The vessels and nerves are just to the inner side of our field: the pectoralis minor muscle crosses them. There is the short head of the biceps muscle, and below, the coracobrachialis.

We cannot furnish the percentage of sarcomata of the shoulder that has reached the autopsy table without metastases. In perusing the literature upon sarcomata involving the upper end of the femur, however, we found that 40 per cent. of the cases had no demonstrable metastases anywhere in the body at autopsy. This signifies that 40 per cent. were curable as a local lesion, even at the time the patients died, and emphasizes that, even in its superlative stage, if the local lesion had been removed 40 per cent. of the patients would have been cured. If that be true when the patient reaches the dead-house stage of development of a malignant tumor, how much more of a chance of permanent cure is there when the patient is operated upon early! This holds, but to less degree, for many of the malignant lesions, particularly the carcinomata.

In investigating the subject of carcinoma we collected 10,315 cases of autopsies from the foremost pathologic institutes of the world, where specific efforts had been made to determine the presence of metastases at the time of death. Of the total number, 19.7 per cent. had no demonstrable metastases in the lymph-nodes or elsewhere in the body at the time of death. Think of it! One-fifth of the cases presented only local involvement at the time of death. That affords some idea of what may be expected of early operation for malignancy. We have been pessimistic about the cure of malignant disease, because so many of our cases have come to operation too late. Perhaps, in the face of these statistics, our pessimism has been unjustifiable.

Now you see that the supraspinatus, the infraspinatus, the subscapularis, the rhomboidei, the pectoralis minor, and the posterior half of the deltoid muscles are out of commission permanently, but we have not interfered with the nerve-supply of the triceps, so that there will be preserved at least its short heads. Nor has the attachment of the biceps been harmed—there is the long head of the biceps muscle in this forceps: we shall displace it and examine the field. Now we must reconstruct the shoulder attachments.

Let the record show that the sarcoma involved the body of the scapula, and that the tip of the coracoid process fractured as we elevated it and was dissected out independently of the remaining portion.

We are irrigating this field with 5 per cent. phenol so as to carry away any malignant cells that might have been left behind. We next attach this short head of the biceps to the acromial end of the clavicle. Here is the long head of the biceps, which we also fix to the clavicle. Now we put the shoulder back into position, fixing the humerus beneath the clavicle: we have preserved this portion of the capsule for that purpose. There is the trapezius muscle again, which we also attach to the capsule and acromial end of the clavicle. The suspension of the humerus is the all-important factor: you can see how it stays fixed to the clavicle (Fig. 276). We insert a drain.

RECAPITULATION

Let the record show that we made a posterior triangular flap incision and exposed the acromion process and the spine of the scapula; that we then made a semicircular incision around in the direction of the axilla, so as to release and throw down the triangular flap; that this gave an excellent exposure of the operative field; that next the muscular attachments to the vertebral border, the superior and the inferior angles and the axillary border of the scapula, were separated, and the scapula was totally removed; that in the reconstruction the muscles were reattached around the shoulder and the fibrous capsule of the joint was firmly secured to the acromial end of the clavicle, so as to hold the humerus well up; to these structures was fixed the margin of the trapezius muscle, so as to support the shoulder by means of the trapezius; that no effort was made to attach the trapezius to the portion of the deltoid that had been divided; that the axillary structures were not disturbed in the dissection; and, finally, that the arm was dressed in a sling and thus supported beneath the elbow.

POSTOPERATIVE TREATMENT

After closure there is but little that would indicate the extent to which the dissection had been carried (Fig. 277). Immediately after the operation the patient will have severe pain, requiring $\frac{1}{10}$ grain of heroin. We shall begin the sodium cacodylate and x-ray treatments on the fifth day, and the Coley serum about the fifteenth. Immediately after the operation the patient will also be given saline clysters, with 20 grains of the lactate of calcium added to each of the first three enemas: this drug supplies the calcium salts which are so essential to the formation of a blood-clot.

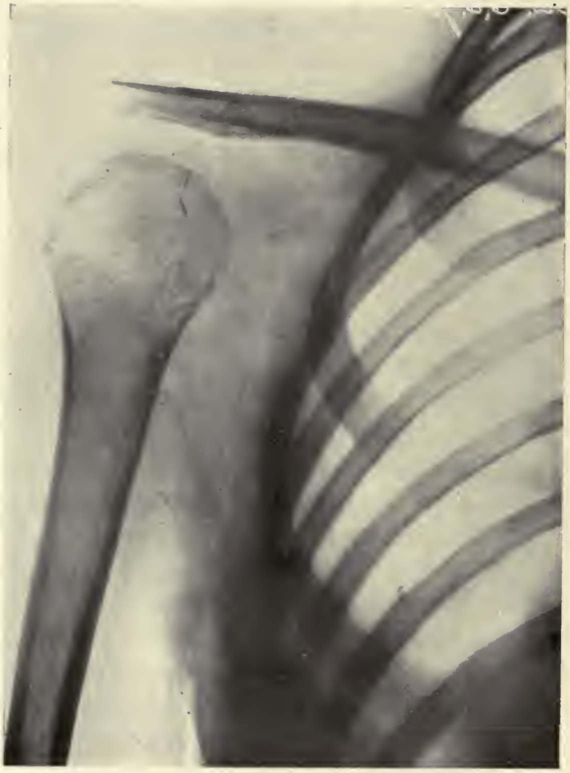

Fig. 276.—Postoperative skiagram showing orientation of bones composing skeleton of shoulder after total excision of scapula for osteosarcoma. Note close juxtaposition of head of humerus to acromial end of clavicle—a juxtaposition obtained by suspending the humerus to the clavicle through suturing of the regional ligamentous structures—posteroanterior view (see under recapitulation).

The pathologist, Dr. Sweek, reports the tumor as a myeloid giant-cell sarcoma with a large number of giant-cells—from three to fourteen to a $\frac{1}{6}$ microscopic field. It appears to be of a rapidly growing type (the exception for the giant-cell variety), and is surrounded by but little connective tissue. The stroma exhibits the spindle-cell type of sarcoma.

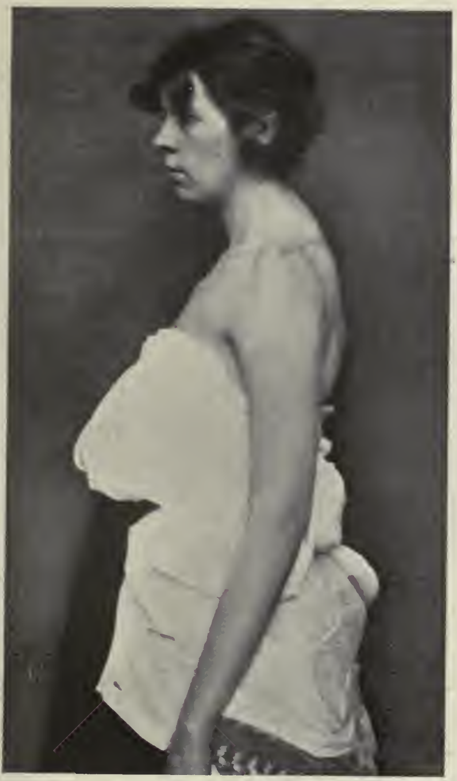

Fig. 277.—Scar two months after total excision of scapula. But little remains to indicate extent to which operative field had been developed.

[*N. B.* (February 5, 1915).—Up to the present time, three weeks after operation, the patient has made an uneventful recovery.

March 14, 1915: Two months after operation, despite the extensive procedure, there is good range of motion of the arm (Figs. 278 and 279).]

[A case of sarcoma of the scapula with resection of the scapula was reported in the Clinics for June, 1913, p. 521. The operation was performed on October 8, 1912, nine months after the first manifestations of symptoms or signs of the malady. A memorandum made on March 1, 1913, at the time of reëxamination six months after the operation, reads: "There is no evidence

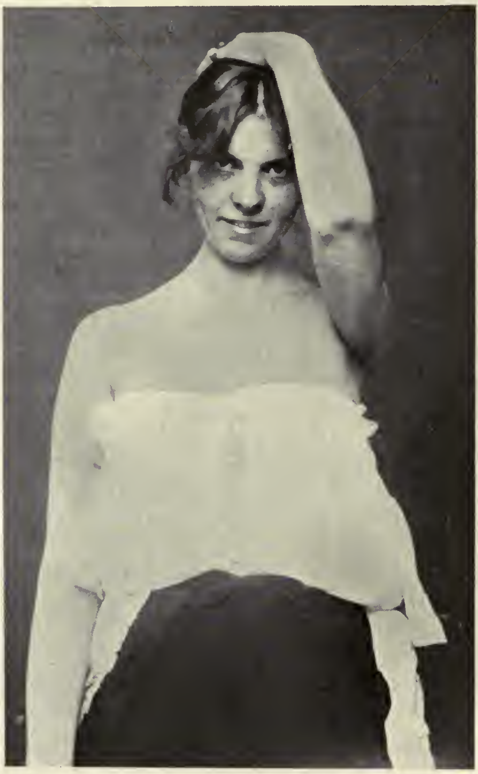

Fig. 278.—Shows extent of voluntary flexion of arm two months after total excision of left scapula. The patient can dress the hair with this arm without difficulty.

of recurrence now, and we hope that there will be none." Unfortunately, our hopes were shattered when the patient died three months later from the effects of pulmonary metastases.]

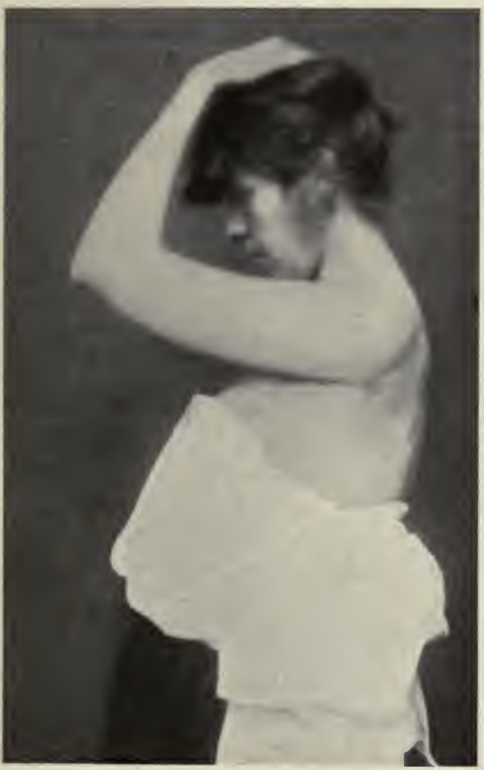

Fig. 279.—Same as Fig. 278 (lateral view). Note depression left after total excision of scapula.

We append a table showing the site-incidence of 797 sarcomata of bone: the scapula was involved 23 times. The table has been compiled from the writings of Polosson and Bérard, of Gask and of Maybury.

TABLE SHOWING SITE-INCIDENCE OF 797 SARCOMATA OF BONE

Head	23	
Jaw	227	
		250
Thorax	20	
		20
Scapula	23	
Clavicle	3	
Humerus	90	
Radius	40	
Ulna	15	
Hand	19	
		190
Ilium	17	
Sacrum	6	
Femur	160	
Tibia	100	
Fibula	25	
Foot	29	
		337
Total		797

OSTEOSARCOMA OF HUMERUS, RECURRENT —INTERSCAPULOTHORACIC AMPUTATION

Summary: Clinical history of patient; comments—at first, resection is the operation of choice, with amputation later if necessary—reasons and illustrative cases; importance of early diagnosis of sarcoma by history and skiagrams; sarcoma locally a circumscribed lesion; resection first affords chance of saving limb; insertion of transplant; steps of operation—method of dealing with vessels and with nerves—muscles removed except latissimus dorsi; points emphasized during operation—path by which sarcoma travels; value of Dr. Senn's experiments; advantage of obtaining *endarteritis traumatica;* nerve-stumps sutured to prevent neuroma-formation; Thoma's positive chemotactic theory for axons; edema of tissues from proximal block of circulation; free bleeding of flap a welcome sign; postoperative comments—*x*-ray, arsenic, and serum therapeusis.

CLINICAL HISTORY OF PATIENT

(Abstracted from the complete report of the case in the CLINICS for August, 1914, pp. 783–803.)

MALE, aged thirty-eight years, admitted May 15, 1914. There were three separate histories of trauma to the right shoulder. The first occurred September 15, 1913. While unloading grain from a freight-car the patient, who was standing upon the platform, strained his shoulder trying to catch a bushel sack of wheat thrown at an unfavorable angle. *The sack did not strike the shoulder.* This injury was followed by a little stiffness and limitation of motion, but no pain.

The second trauma occurred three weeks later, October 8, 1913. While walking he caught one foot in a cable and again strained the right shoulder by throwing both hands back to regain balance. Great pain followed this injury.

The third trauma occurred five weeks before admission, April 8, 1914, when the patient removed his coat and swung it over the back of a seat with the injured arm. First a snap and then a severe pain were felt. Since then the upper portion of the right arm has rapidly become enlarged (Fig. 280).

The arm has been manipulated freely by many kinds of practitioners.

Dr. Murphy's interpretation was that sarcoma of the humerus resulted from the first trauma, and pathologic fracture (Fig. 281) from the third, and that the undue manipulations had played a rôle in accelerating the growth of the neoplasm.

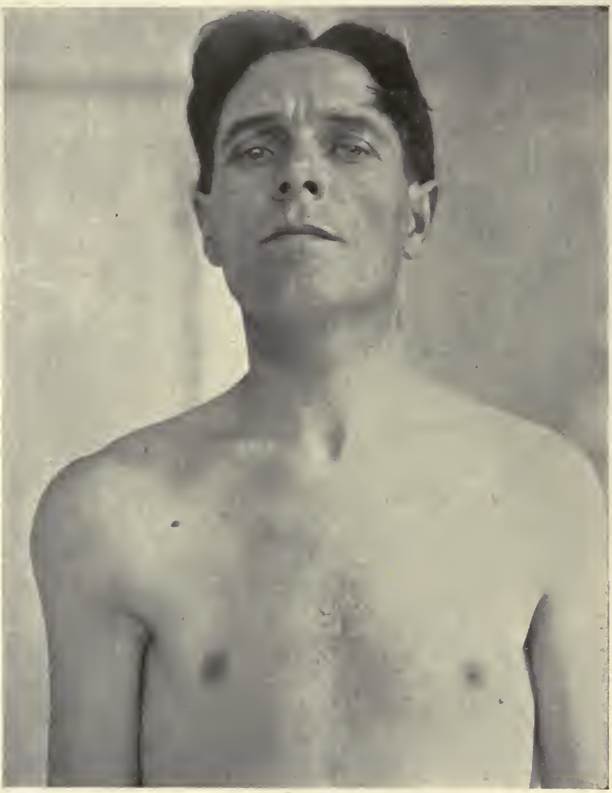

Fig. 280.—Osteosarcoma of upper end of right humerus. Note bulging of upper portion of deltoid region of arm. Photographed before the first operation, on May 15, 1914, four months previously.

At the first operation, May 15, 1914, the upper end of the humerus, bearing the sarcoma, was resected (Figs. 282, 283, 284).

At the second operation, June 29, 1914, it was planned to insert a tibial transplant into the humerus, but the presence of a local recurrence beneath the deltoid muscle led to abandonment of that plan.

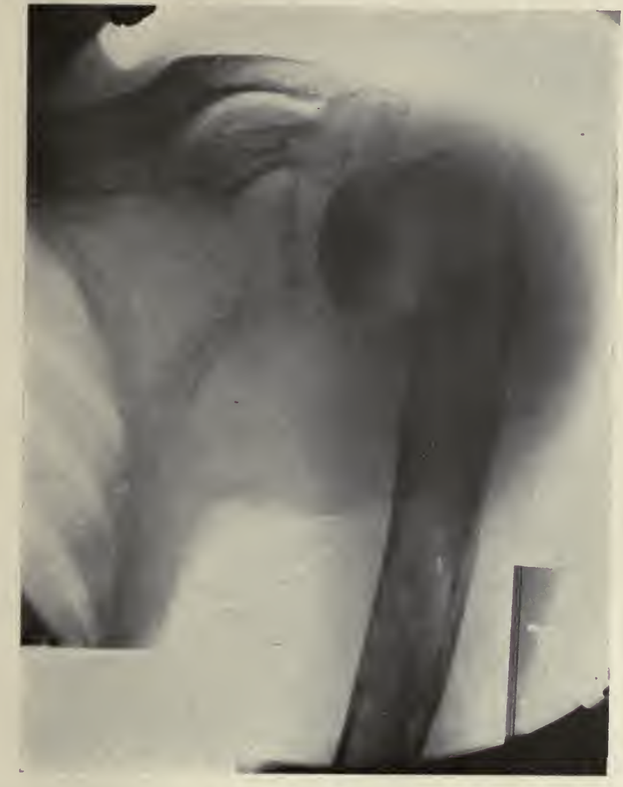

Fig. 281.—Skiagram showing osteosarcoma of upper end of right humerus as it appeared just before the time of the first operation, on May 15, 1914, four months previously. Note pathologic fracture of surgical neck of humerus with external displacement of upper end of lower fragment, accounting, together with the bulk of the tumor, for the subdeltoid bulging shown in Fig. 280 (posteroanterior view).

Since the second operation there have been continual pain and rapid growth of the tumor. The axilla is well filled with the mass.

COMMENTS

Dr. Murphy (September 9, 1914): Previous to the time of the first admission, May 15, 1914, the patient had been subjected to various kinds of treatment. His arm had been massaged and manipulated by a chiropractor, a neuropath, and an osteopath. The history was a definite one of an osteosarcoma with pathologic fracture. At that time we were doubtful of the presence of internal metastases. After resection of the upper

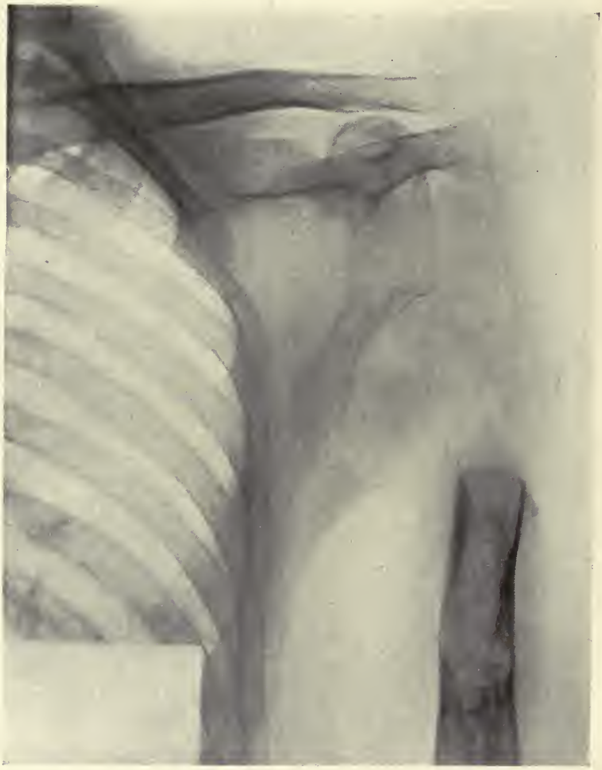

Fig. 282.—Skiagram of right shoulder taken two months after resection of upper third of humerus for osteosarcoma. Note recurrence of the malignant growth in the humerus. This recurrence, which involved the soft tissues about the shoulder as well, established the indication for the present operation of interscapulothoracic amputation (posteroanterior view).

extremity of the humerus bearing the whole neoplasm, we ordered large doses of arsenic and of x-rays, and since then have watched him carefully. Upon examination of the lungs and other portions of the body we found no evidence of metastases. Now we intend performing interscapulothoracic amputation. We did not insert a transplant because of doubts concerning the local as well as the constitutional condition. The plan was to make a transplant from the tibia, provided no local recurrence would ensue.

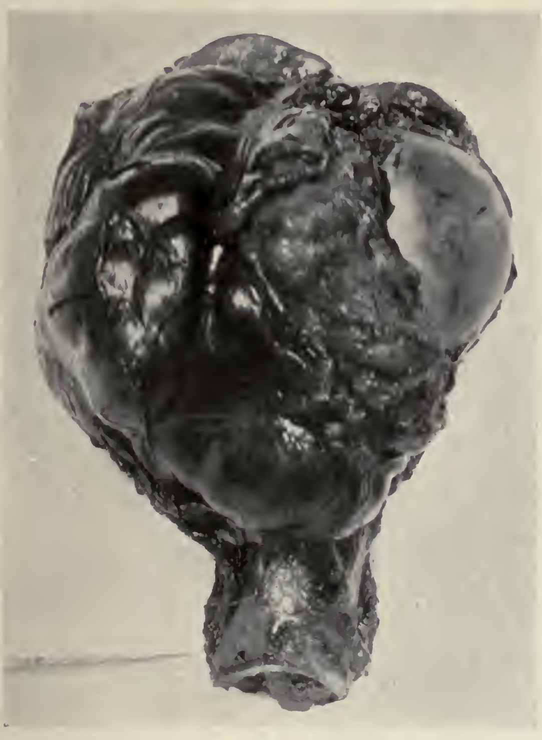

Fig. 283.—Operative specimen of upper third of humerus involved in osteosarcoma. Note position of articular surface of head of humerus (anterior view, taken after first operation).

We have a patient, a girl, to show you to-day. It is now two years and nine months since we resected the upper extremity of the right tibia for sarcoma and removed a transplant from the left tibia to bridge the resulting gap. She has had no recurrence.

We believe that in these cases of sarcoma the diagnosis can and should be made early, by the history, and with the aid of skiagrams. We believe also that sarcomata are circumscribed lesions in so far as the local focus is concerned, and therefore that the latter can be removed as well by resection of the bone involved as by amputation. Recurrence has taken place in the muscle, but not in the bone below, so that by primary resection the patient has had all the advantages to be derived from amputation at the shoulder-joint without the disadvantage of immediate loss of the upper extremity. The disadvantage now is the necessity of another operation.

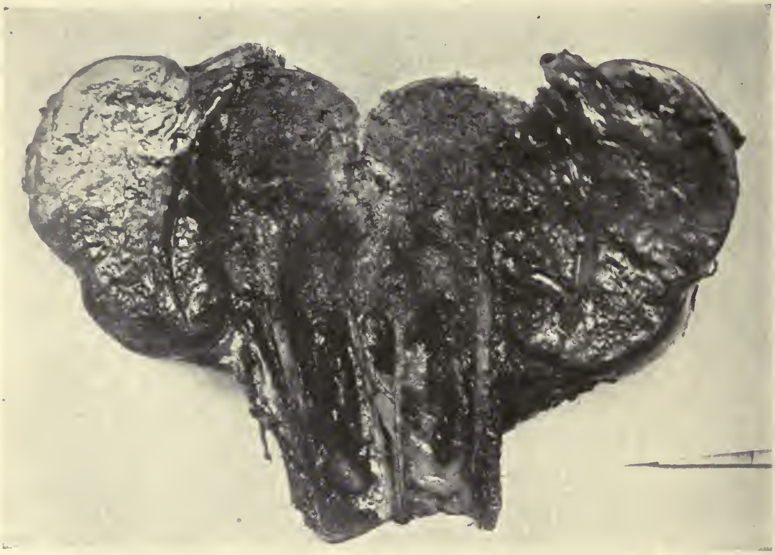

Fig. 284.—Operative specimen of upper third of humerus involved in osteosarcoma. Specimen split open to show breech in capsule of sarcoma, invasion of medullary cavity of humerus by sarcoma, and pathologic fracture of surgical neck (photograph taken after first operation).

The removal of the upper end of the humerus is not difficult. Furthermore, the stumps of the various muscles of the shoulder can be brought together and united as one central mass, both in resection and in insertion of a transplant. In the latter case the muscles may be stitched around the transplant at once, for tuberosities develop subsequently, and the muscles soon become reattached. We have as a patient a girl of seventeen who was operated upon five years ago for sarcoma of the humerus: we resected a little more than the upper half of the bone and reattached the muscles; she can use that arm now just as well as its fellow.

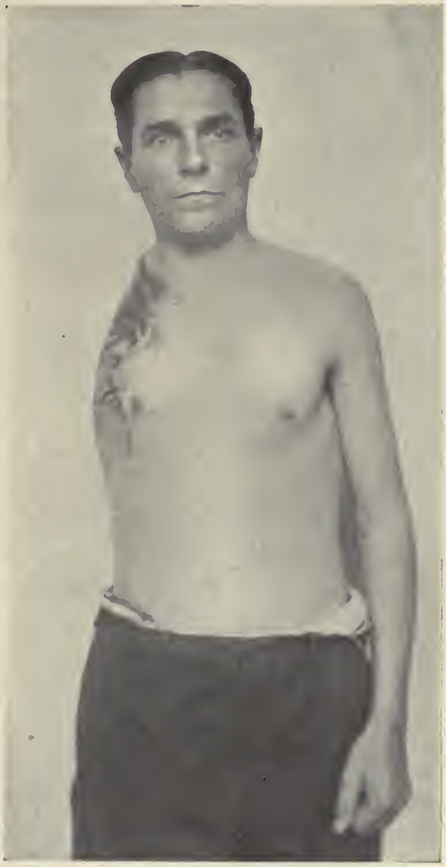

Fig. 285.—Operative scar after interscapulothoracic amputation for recurrent osteosarcoma of right humerus.

OPERATION

[Dr. Murphy began the operation by dividing, after incision and exposure, the pectoralis major and minor muscles from their costal attachments, the former close to the sternum; after elevating these muscles he exposed the axillary artery just as it emerges from beneath the clavicle, and ligated it with a double catgut ligature. After the ligature had been applied the artery was clamped proximal to the site of the proximal ligature to traumatize the intima, favor clot-formation, and afford a double means of security; a double ligature was also placed around the vein; then he blocked each nerve-trunk of the brachial plexus with three drops of a 0.5 per cent. solution of novocain, and to prevent neuroma-formation after resection of the plexus he stitched together end-to-end the stumps of the nerve-trunks. Then the clavicle was divided with the bone-forceps, and the upper extremity, including the scapula, was removed; all muscular origins from the chest were separated except that of the latissimus dorsi, which was divided about three inches from the humerus and used to support the flap, and this was covered by the skin from over the scapula. The wound was closed with drainage (Fig. 285).]

POINTS EMPHASIZED DURING OPERATION

Sarcoma does not travel through the lymphatics: it travels through the cellular interstices of the muscles, arteries, and bones.

We tie the ligatures one-half inch apart: that is what we learned from the late Dr. Nicholas Senn in the experimental work which he performed at Milwaukee under very unfavorable conditions, but which has been of such immense value to the profession.

We traumatized the axillary artery by clamping it proximal to the point where it is ligated, so as to start up *endarteritis traumatica* and accelerate thrombosis of the vessel. This we learned from our experimental work in arterial suture in 1896. (Murphy: "Resection of Arteries and Veins Injured in Continuity—End-to-End Suture. Experimental and Clinical R search," *Medical Record*, January 16, 1897.)

Shock was avoided by paying attention to and checking hemorrhage from the small vessels while the limb was being amputated. It was also avoided by novocainizing the freshly severed ends of each nerve-trunk: although cut, yet the axons of the peripheral portion still live, and would have continued responding to stimuli for eighteen hours.

We suture these nerve-ends to prevent neuroma-formation and consequent neuralgias that are worse than death: in doing this we direct the axons toward each other. According to Thoma, there is a *quasi* positive chemotactic attraction which draws these axons toward each other. In some experiments on the nerves the ends faced in a contrary direction, but turned back and united to their trunk-axons on the opposite side.

There is edema of all tissues on account of the blocking of the circulation on the proximal side.

The upper flap oozes freely, and we enjoy seeing it ooze, because it assures us of the vitality of the flap.

POSTOPERATIVE COMMENTS

1. *X-ray Therapeusis.*—Early after the second operation the tumor diminished under arsenic and *x*-ray therapeusis, but ultimately the tumor began to enlarge, as is usual with *x*-ray, arsenic, and radium treatments. The tumors do not respond a second time: they respond the first time to radium, to *x*-ray, and often to arsenic, but once they begin to grow again there is no further response to these measures.

VISITING PHYSICIAN: Why do you use the *x*-rays now after this operation?

DR. MURPHY: We use them to destroy any cells that have escaped our operative dragnet. It is fairly well established that often malignant cells, both sarcomatous and carcinomatous, are isolated *in loco* by connective-tissue encapsulation: *x*-rays are used merely to stimulate connective-tissue reproduction.

2. *Arsenic Therapeusis.*—We give arsenic in the form of the cacodylate of soda, and in large doses of from 10 to 12 grains. (See CLINICS, October, 1915, pp. 953–954.) For many years it has been known that arsenic arrests the growth of certain neoplasms that histologically are sarcomatous, but from the clinical history, benign.

After the operation the arsenic and *x*-ray treatments will be continued.

3. *Serum Therapeusis.*—VISITING PHYSICIAN: What has been your experience with Coley's serum?

DR. MURPHY: My experience has not been good; in fact, it has been very bad. All the cases have terminated fatally, including those I sent to Dr. Coley himself. From Coley's statements, which I believe are in consonance with the facts, some of the patients get well, undoubtedly, and for some months our patients have appeared to do remarkably well under this treatment, but the process later took on renewed activity and then did not respond markedly to a second treatment.

[*N. B.*—This patient died some six months later from metastatic sarcoma of the lung.]

[Of comparative interest is the case of "Osteitis Fibrosa Cystica of Humerus," which was published in the Clinics for October, 1912, p. 741.]

CICATRICIAL FIXATION OF ULNAR NERVE IN ITS GROOVE SEQUENTIAL TO ANCIENT FRACTURE OF OLECRANON PROCESS—RELEASE AND TRANSFERENCE OF NERVE TO NEW SITE—RESECTION OF OLECRANON-TIP

Summary: Clinical history of patient; comments—cause of trophic changes in hand and fingers; stretching of nerves without separation a more serious injury than direct division; operation—exposure of ulnar nerve behind elbow; nerve congested and thickened, forming a neuroma, and fixed by a cicatricial band; nerve released from band and faradic excitor applied to determine its conductivity; ulnar nerve lifted from its groove; resection of tip of olecranon process; triceps tendon found adherent to periosteum of humerus—released—interposition of fascia-fat flap to prevent re-adhesion; ulnar nerve secured permanently in front of internal epicondyle by suturing fat and deep fascia over it; recapitulation; postoperative comments. Case of late paralysis of ulnar nerve following fracture of external condyle of humerus.

CLINICAL HISTORY OF PATIENT

The patient, a male aged forty-six years, was admitted to hospital May 17, 1915. The chief complaints were the inability to flex or extend the right forearm beyond the range of a few degrees; inability to supinate the forearm; hyperesthesia of the ring and little fingers, and discoloration of the tip of the little finger.

The present condition is the result of an injury that was received fourteen months previously, on March 26, 1914. While the patient was riding in a buggy and leading a horse behind with the right hand, the horse he was leading became frightened and bolted, at the same time pulling him out of the buggy. He struck with the ulnar side of the right forearm and the outer side of the right elbow against the pavement. Immediately after the injury the ring and little fingers became numb, and the forearm pained along the ulnar side from elbow to hand. A physician applied a circular gypsum case and dressed the forearm between full extension and right-angle flexion. The gypsum case was applied at 9 A. M. on March 26th, and one hour later the forearm began to swell and ache intensely. The hand became swollen and bluish in color. The forearm ached the entire day, and by night-time the pain had become so severe that he had to summon the physician, who, upon his arrival, cut the gypsum case and then applied a muslin roller bandage around the outside of the gypsum case. After the gypsum case had been cut the patient felt much relieved and the bluish color of the hand disappeared: the swelling in the hand persisted, however.

The gypsum case was left on three months, and during that time the ring and little fingers felt numb and an occasional twinge of pain shot down the ulnar side of the forearm to the little finger. After it was removed there was no motion in the elbow. The patient, therefore, consulted another physician, who administered an anesthetic and reset the elbow-joint on July 13th. The arm was dressed on a splint in the position of extension: this splint was left on four days. For three months after the removal of the splint the forearm was manipulated, but without improvement. In November an anesthetic was again administered and the forearm reset and dressed in the position of complete flexion. Since then the elbow-joint has been manipulated, but without success, and the forearm has felt numb and weak.

On February 1, 1915, over three months previous to admission, the patient noticed a small water-blister at the tip of the little finger: this blister was opened and serum escaped. The finger did not heal, and within a few days became bluish in color and finally black: since then it has been treated with desiccating-powder dressings.

At present the patient is able to flex and extend the elbow-joint through a range of only 10 degrees. He cannot supinate but can pronate the forearm within a limited range: he cannot extend the fingers fully. There is constantly a sensation of numbness in the little finger and in the ulnar side of the ring-finger. The patient occasionally has shooting pains from elbow to little finger along the ulnar side of the forearm.

Physical Examination.—On palpation there appears to be a neuroma on the ulnar nerve, which is very sensitive from a definite point just on a level with the internal epicondyle of the humerus. Every time I touch the neuroma it causes pain: this local sensitiveness is due either to the original injury, from stretching of the nerve with intraneural deposits, or to a partial division of the nerve. The area supplied by the ulnar nerve is very sensitive superficially. The three fingers supplied by the median and radial nerves do not pain at all, although they are stiffer than they should be. The radial pulse is normal. There was no injury to the clavicle, of which the patient is aware. A traction injury with stretching of the whole brachial plexus (CLINICS, February, 1915, p. 77) may account for the nerve-signs in his case, because the entire hand is edematous.

There is a mass above the olecranon process and the olecranon seems to incline backward, but there is a mass also a little distance from the olecranon: it looks as though it were in the triceps muscle; this mass does not show in the skiagram.

Trophic changes exist in the hand from an irregular line just above the wrist, showing that there is a disturbance of the nerve-supply resembling that which one finds when a cervical rib disturbs the plexus (*vide supra*, p. 1069), and in certain cases of Raynaud's disease. On the right side there is atrophy of the deltoid muscle: this could be the sequence of disuse. The shrinkage of the muscles in the distribution of the ulnar nerve is pronounced.

COMMENTS

DR. MURPHY (May 22, 1915): Ten months after the accident the patient began to show signs of trophic changes in the hand: that these trophic changes were not due to circulatory disturbances is indicated by the full and vigorous pulsation of both the radial and ulnar arteries; in fact, there has been no etiologic factor to disturb the circulation. But why should trophic changes in the hand and gangrene of the tip of the little finger have appeared at that time? It is this point which I want to discuss with you.

The case is very extraordinary, and the outstanding features are these: First, the nature of the accident: it was a pulling accident. The patient was seated in a buggy, driving with the left hand and leading a horse behind the buggy with the right hand. The horse became frightened: he kept hold of the rope with which he was leading the horse, and the horse's pull on the rope twisted the right arm, subjected the brachial plexus to traction, and eventually drew him out of the carriage. He fell upon the pavement, striking upon the elbow, fractured the olecranon, and injured the shoulder. Ten months later trophic changes appeared in the hand, ring and little fingers.

That hand looks just as a foot looks preceding diabetic gangrene: it has the same bluish-purple tint. These trophic changes are appearing not because the blood-vessels have been injured; not because the olecranon process has been fractured; not because the shoulder has been injured: but because the brachial plexus has been stretched. Such a stretching without separation of the axons is one of the worst forms of injury to nerves—it is many times worse than direct division. When I look back over past events it seems miraculous that we did not have more of these trophic changes in the days when we stretched nerves for the relief of tetanus; and it seems miraculous that we did not destroy nerves we used to stretch to relieve neuralgia by putting a No. 30 male urethral sound under a nerve and then working the sound up and down vigorously. After such manipulations as these the patient might have exhibited temporary paralysis in the distribution of the nerve involved, but motion always returned eventually.

The first thing I want to do to-day is overcome the fixation in the shoulder. This is the type of case that Mr. Robert Jones, of Liverpool, relieves by forced manipulations without anesthesia; in my opinion it is unnecessary to cause such intense pain. I am now freeing the capsular adhesions in the shoulder that sprung from the traumatic synovitis which was a sequence of the injury.

Had the cause of the trophic changes been arterial from a distinct trauma to the vessels, the gangrene would have appeared within two, three, or four days after the injury; but since the trophic changes did not appear until ten months after the injury, they must have resulted from the stretching of the axons and the formation of cicatricial tissue in the lacerated nerve: it is, then, the contraction of this cicatricial tissue in the nerve which accounts for the present hyperesthesia. The slightest touch of any object gives rise to severe pain; not only has the ulnar nerve been involved in the stretching process, but the brachial plexus as well: the circumflex nerve shows that it is involved by the atrophy of the deltoid muscle, and there are trophic changes in the zone of the hand supplied by the median nerve—changes which all show that the brachial plexus has been affected.

OPERATION

Incision made to expose ulnar nerve.] There is some thickening of the ulnar nerve just behind the elbow, and the nerve is fixed by a mass of cicatricial tissue which formed about the site of the fracture of the olecranon process. The question arises as to whether the nerve is the seat of compression or of partial division with an effort at fibrous repair. I must expose the ulnar nerve for its entire course in its relation with the elbow. There is the area of thickening about the internal condyle of the humerus: here is a band of cicatricial tissue fixing the nerve. . . You see me sever the band and expose the ulnar nerve: I want you to notice the condition of the nerve (Fig. 286). First observe the pathologic vascularization of the nerve, resembling that shown in the pictures from the Frankfort Pathologic Institute. Above the site of fixation the nerve appears to be normal: it is white in color and cleancut in outline. Here, where it is fixed in its groove between the olecranon process and the internal epicondyle, the nerve is altered in color (pinkish) and in shape (bulbous). You have merely to glance at one of these fixed nerves to remember it and have its pathology a part of your knowledge. It is this bulbous, fixed portion that gave rise to the neuritis and the hyperesthesia. The nerve, however, is not out of commission entirely, but shows that it is subjected to continual irritation during the motions of flexion and extension. We must shift this nerve from the zone of fixation to a fixation-free

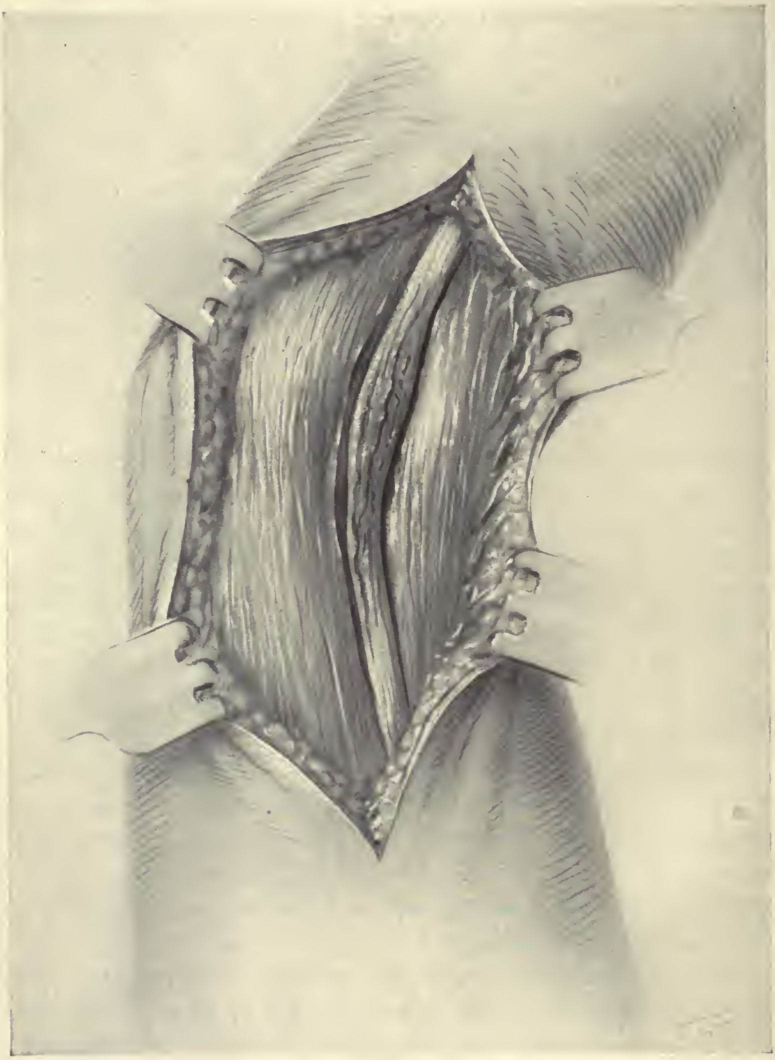

Fig. 286.—Showing the enlargement and vascularization of the ulnar nerve where it was repeatedly exposed to trauma during flexion and extension of the elbow-joint, while fixed between the constricting band and the bony floor of the ulnar groove. Right arm. Sketch made at operating table with arm in the operative position of elevation (postero-internal view).

zone: we cannot let it remain here where it will be exposed to further trauma by the fixation and continual irritation. In all my elbow-joint arthroplasties I first shift the ulnar nerve from its groove to a new bed remote from contact with the bone. . . . There is the neuroma: it is the sequence of the repeated trauma and the continual stretching. This patient requested me to amputate his arm for fear of the gangrene spreading up the limb from the gangrenous little finger.

To determine the integrity of nerve-conduction we may apply two tests. One is applied to the nerve distally to see whether it is living below the lesion at the elbow and whether it responds to the faradic current by contracting the muscles. [Applies faradic electric excitor to nerve distal to neuroma.] The flexor carpi ulnaris responds: now we know that the distal axons are living, for otherwise there would be no faradic positive reaction. Remember, a dead nerve does not respond to the faradic current. Now let us see if the faradic current passes through the connective tissue at the site of the neuroma. [Applies excitor to neuroma.] . . . The current passes directly through the neuroma all right, showing not only that the nerve-filaments distal to the neuroma are living, but also that at the site of the neuroma they have been regenerated despite the presence of the connective tissue, and that all they need is to be released from the fixation-band and then be given time to regenerate further. . . . I am retracting this flexor muscle in order that I may lift the ulnar nerve out of its groove and shift it into a new bed away from bone.

We shall now turn our attention to the fractured tip of the olecranon process. When the patient attempts to extend the forearm, the olecranon process in its ascent strikes against the olecranon fossa, which here is filled in with a dense fibrous mass. I must resect this offending tip of the olecranon process. See! I have succeeded in removing the tip without traumatizing the elbow-joint.

The next step is to prevent the tendon of the triceps muscle from uniting to the periosteum of the humerus, to which we found it fixed by the upward extension of the fibrous mass filling in the olecranon fossa of the humerus: otherwise there will again be a firm union of this triceps tendon to the periosteum. [Dissects

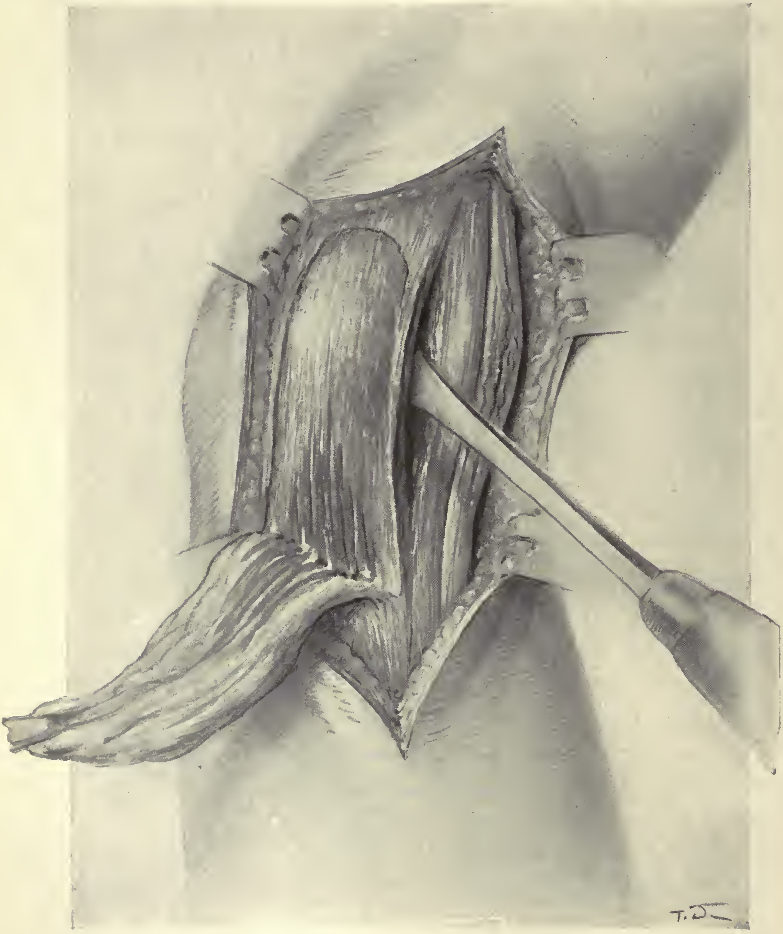

Fig. 287.—Showing the ulnar nerve released from its constricted position in the ulnar groove and displaced forward in front of the internal epicondyle, and the preparation of the flap from the triceps sheath and the fat covering it, pedicle upward, for insertion between the triceps tendon and the periosteum of the humerus. Artist's chisel engaged in exposing tip of olecranon process for resection.

flap from fascia and fat covering triceps tendon (Fig. 287).] This was the way I originally prepared the flaps for the operation

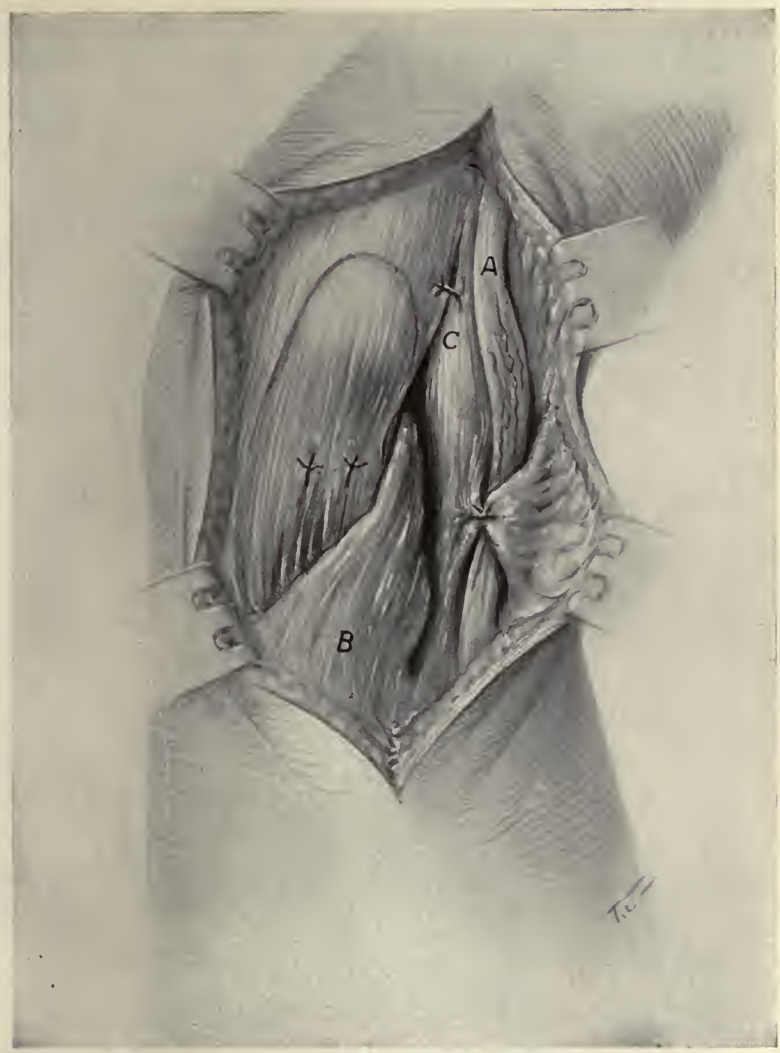

Fig. 288.—*A*, The ulnar nerve displaced permanently to a position in front of the internal epicondyle of the humerus. *B*, The pedicle of the fascia-fat flap; the tongue end is swung around the inner border of the triceps tendon to a position between the tendon and resected olecranon-tip and the periosteum of the humerus. Two tacking sutures are seen passing through the triceps tendon to anchor the tongue in place. The fat from the inner edge of the incision is sutured over the ulnar nerve in its new bed to the lower portion of the fibrous internal intermuscular septum, thereby preventing the ulnar nerve from slipping back over the internal epicondyle, *C*, to its normal position behind that bony process.

of arthroplasty of the elbow, except that I had the base in the opposite direction—that is, I put the base downward instead of upward, as in this case (Fig. 287). See how I insert this flap in front of the triceps tendon so that it will not become adherent

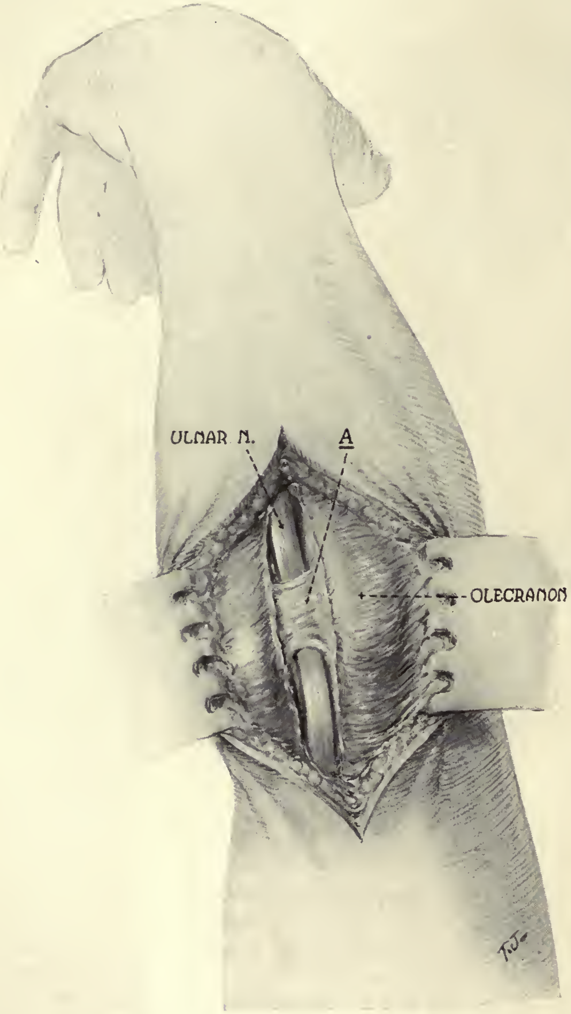

Fig. 289.—Sketch from another patient of a condition similar to that described in text. At *A* there is shown a cicatricial band fixing ulnar nerve in its groove, which is due to an ancient fracture of the olecranon-tip. Sketch made at operating table, with arm in operative position of elevation (postero-internal view).

to the periosteum of the humerus again (Fig. 288, *B*)! We keep the ulnar nerve permanently displaced from its groove so that it cannot possibly get back in again. . . . Now I am

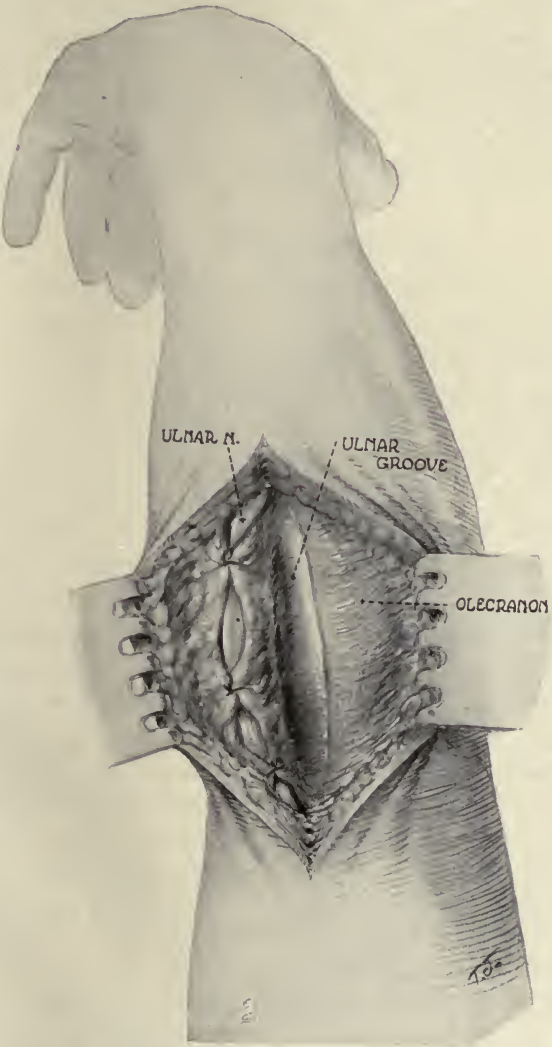

Fig. 290.—Sketch showing cicatricial band removed, and ulnar nerve transferred to a tension-free position in front of internal epicondyle, and secured in place by fascia-fat flaps. The olecranon-tip was removed as the next step in the operation.

fixing the ulnar nerve in the position in which I wish to retain it, namely, in *front* of the internal epicondyle (Fig. 288, *A*), entirely out of the field, and retain it there with fat and fascia, so that it cannot come back into the field. . . . It is now fixed permanently. Now we wish to fix this olecranon process by covering its resected tip with a flap of fascia so as to prevent a growth of bone on its denuded surface. You see it is easily accomplished. The tension should be taken off of the skin stitches by placing 3 or 4 catgut stitches in the subcutaneous fascia.

RECAPITULATION

Let the record show that upon exposing the ulnar nerve we found the neuroma that we felt through the skin before the operation; that positive faradic tests showed, in consonance with our interpretation of the symptoms, that there was no division of the nerve; that there was pathologic vascularization and an enlargement of the nerve to twice its normal diameter for a distance of 1¾ inches behind the internal epicondyle, while above this point the nerve was not congested, but presented the normal white tint; that the ulnar nerve was freed entirely from the fixation band and displaced forward from its groove; that the olecranon fossa was filled up by a dense, fibrous mass against which the tip of the olecranon process struck; that while resecting the tip of the olecranon process it was found that there were adhesions between the triceps tendon and the periosteum of the humerus, these adhesions constituting the point of greatest fixation, as well as the greatest hindrance to flexion; and finally that the adhesions were dissevered and a flap of fat-bearing fascia of the triceps with pedicle above was prepared and inserted in between the triceps tendon and the periosteum of the humerus.

POSTOPERATIVE COMMENTS

The elbow-joint will be dressed at right angles for a few days and then put slowly through passive motion, but never moving it passively to the degree that produces severe pain.

In a paper entitled "An Undescribed Ulnar Nerve Trouble Due to Tension from Scar, and its Cure," F. J. Cotton (*Bost.*

Med. and Surg. Jour., vol. clxxii, No. 13, pp. 480–483, April 1, 1915) reported seven cases closely similar to the above. He writes: "The mechanism is purely *fixation of the nerve* by scar tissue at the turn of the elbow, where it lies in the epitrochlear groove, or in the inch next below this point. Given a nerve so fixed, it must necessarily happen that flexion of the elbow must pull upon it, and ordinary use of the arm must give *intermittent traction* on the nerve-trunk. . . . Relief of this condition is surgical—the only remedy is removal of the nerve to a more favorable site. The conditions found at operation . . . lead me to class these as lesions due to the constant irritation of the fixed nerve by flexion and extension movements, rather than as compression lesions. . . . It behooves us to keep an eye open for these cases—if for no other reason, because they are so easily cured and so quickly."

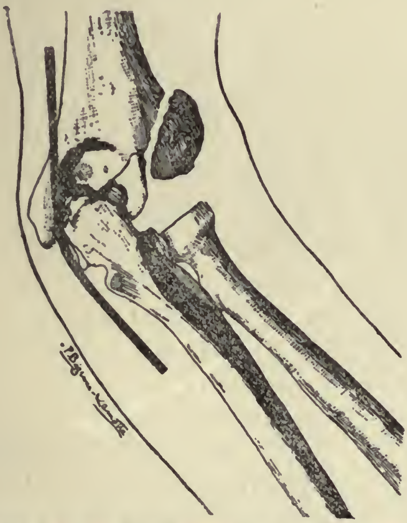

Fig. 291.—Diagram showing the tension of the ulnar nerve over the internal edge of the olecranon in the cubitus valgus following fractures of the external condyle (Mouchet).

The accompanying sketch (Fig. 291), which is from an article by Mouchet entitled, "Paralysies tardives du nerf cubital á la suite des fractures du condyle externe de l'humérus" (*J. de chir.*, 1914, xii, 437—from *Intern. Abstr. of Surg.*, October, 1914, 375) will be found of interest in connection with the above case.

Mouchet has had 7 cases of paralysis of the ulnar nerve coming on at periods varying from five to twenty-seven years after a fracture of the external condyle of the humerus. In all the cases there had been a fracture of the external condyle in infancy or early childhood. The ascent of the external condyle and the change in the line of articulation alter the position of the olecranon, bringing it nearer to the internal epicondyle, so that the ulnar groove is partially obliterated and the nerve is more or less stretched over the inner border of the olecranon, especially when the elbow is extended. Also because of the changes in position there is an exaggeration of the physiologic carrying angle (*cubitus valgus*). Finally, as a result of the abnormal position and the tension on the nerve, neuritis develops. The fracture may have occurred so long before that the patient has forgotten it, and various mistaken diagnoses are made. *Whenever there are symptoms of neuritis of the ulnar nerve and the cause is not known, an examination should be made for evidence of an old trauma.* (Italics ours.) The author treats these cases by supracondyloid cuneiform osteotomy of the humerus.

[Of interest in connection with the above is the case of "Neuroma of the Ulnar Nerve, Result of Cicatricial Compression Following Unrecognized Fracture of the Internal Epicondyle of the Humerus," which was published in the Clinics for April, 1914, pp. 369–374, and a second case occupies pp. 375–380; of additional interest are the cases of compression, laceration, and division of the ulnar nerve, which were published in the Clinics for October, 1915, pp. 823, 833, and 877, respectively.]

HYPERFLEXION FRACTURE OF RADIUS AND ULNA, LOWER THIRD — REDUCTION — BLOODLESS FIXATION BY EXTRA-ARTICULAR NAILING

Summary: Clinical history of patient; comments—hyperextension (Colles's) *vs.* hyperflexion (*not* a Colles's) fracture; factors that indicate operative treatment in this case; the method of fixation by extra-articular bloodless nailing; after reduction of a Colles's fracture internal fixation of fragments is rarely necessary; splints not intended to effect reduction of fragments, but to immobilize them after reduction; operation—reduction of fragments by manipulation; fixation by nails; dressing applied; postoperative comments—nature of deformity; method of reducing a Colles's fracture; tests of reduction; sequelæ of incomplete reduction; operative treatment of ancient, unreduced Colles's fracture; loosening up of tendons in an ancient Colles's fracture of the opposite (right) side.

CLINICAL HISTORY OF PATIENT

The patient, a female aged fifty-eight years, was admitted to hospital May 19, 1915.

Five weeks ago, on April 16, 1915, while stepping from the curb to the street, a distance of six inches, the patient became faint and fell over upon her face; as she was falling she threw forth her hands to break the fall, but struck with the left arm doubled under her. She was picked up immediately, though in a dazed condition, and was conveyed to a hospital.

At the hospital examination revealed a fracture in the left forearm. The left arm felt numb and lifeless. The right wrist was painful. There was a contusion of the left eye and forehead. The left arm was dressed in flexion and a splint applied and kept on until admission into this hospital. The left forearm pained greatly and was somewhat discolored.

COMMENTS

Dr. Murphy (June 2, 1915): When you look at this skiagram (Fig. 293) you see a fracture of the radius and ulna with com-

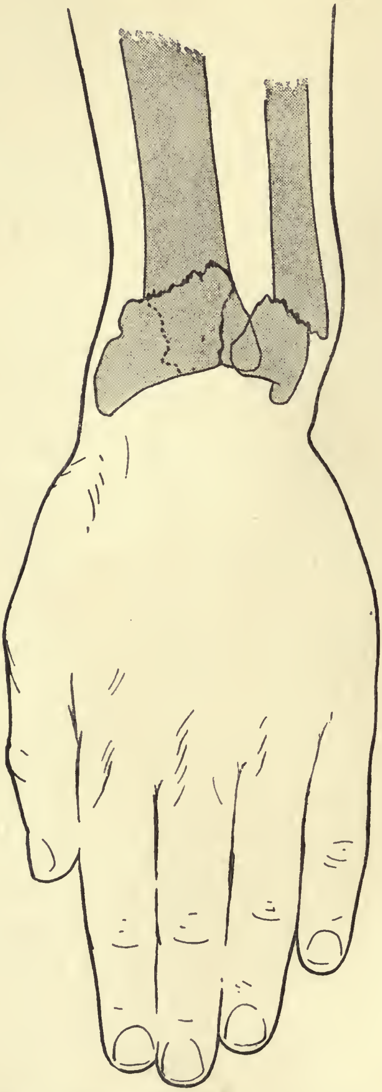

Fig. 292.—Schematic drawing showing extent of displacement of lower fragments to the radial side. Note, in addition, the following features: (1) The comminution of the lower fragment of the radius. (2) The ascent of the tip of the styloid process of the radius to the same level as that of the ulna. (3) The disturbed contour of the soft parts of the wrist, and the inclination of the hand away from the ulnar and toward the radial side (dorsal view).

minution of both bones. Is that a Colles's fracture? No, not at all. A Colles's fracture never results from forced hyperflexion of the hand. The patient states that she tried to put the hand out, but that before she could do it she struck the ground *with the forearm doubled under her:* the result is a fracture of the radius and ulna with volar displacement of the distal fragments (Fig. 295). A *Colles's fracture*, on the other hand, occurs from a fall upon the *outstretched* palm only. Whenever you see a case of fracture just above the wrist, if the patient remembers how he received the impact, then you can decide at once whether the fracture is or is not a Colles's fracture. *A Colles's fracture always results from volar pressure-mechanism: a Colles's fracture* NEVER

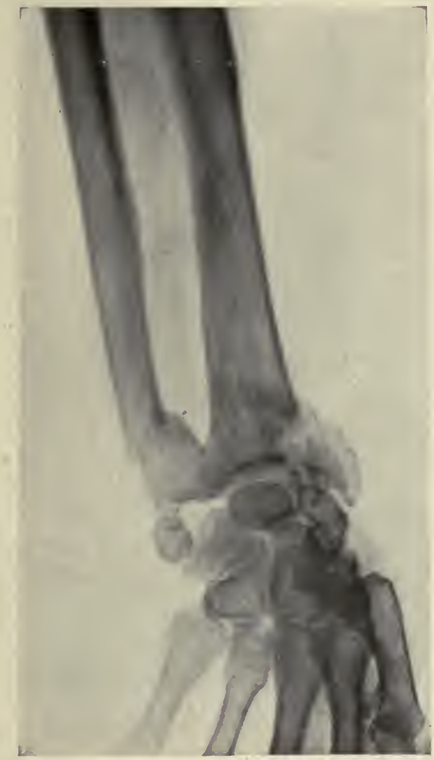

Fig. 293.—Preoperative skiagram showing features accentuated by Fig. 292 (antero-posterior view).

results from dorsal pressure-mechanism. Here is a very pronounced and a very nasty deformity. So far as this first picture is concerned (Fig. 293), it looks very much like a Colles's fracture, but reference to the lateral view (Fig. 295) shows that it is not a fork-handle deformity. In this patient's fracture the bend does not occur where the bend in a fork-handle occurs: it is a *forward* bend, and in order to have a fork-handle deformity the lower fragment of the radius must be displaced dorsally. Why? Because when the fracture is caused by a mechanism of the

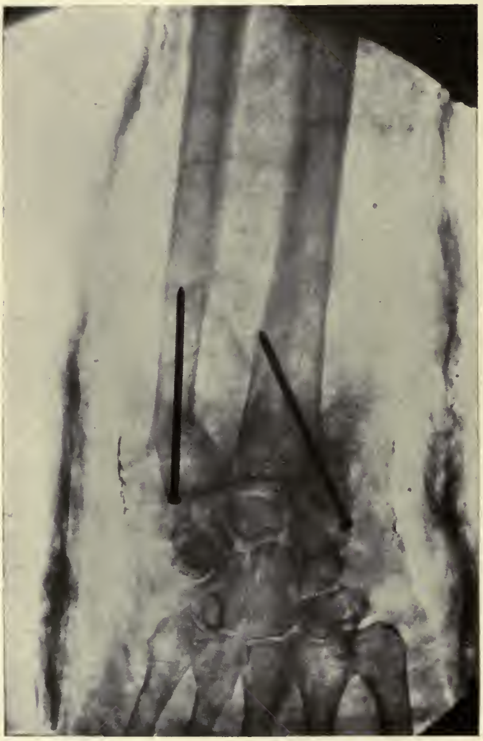

Fig. 294.—Postoperative skiagram showing reduction of fragments and maintenance of reduction by extra-articular fixation with ten-penny "finishing" nails. The point of entrance of each nail is in close relation with the styloid process of its respective bone. Compare with Fig. 293 (anteroposterior view, taken through plaster-of-Paris dressing).

hyperextension type, the plane of fracture involving the radius is always either perpendicular or oblique to the long axis of the radius, and the lower fragment of the radius is displaced dorsally and then upward by the still-unchecked action of the vulnerating force. From this hyperextension-mechanism the fork-handle deformity arises, and this fork-handle deformity is characteristic of the fracture according to Colles's original description, which appeared one hundred and one years ago (*Edinb. Med. and Surg. Jour.*, 1814, x, pp. 182–186).

This fracture is a very unpleasant one to deal with, because of the comminution of the radius and the coincident fracture of the ulna. In consequence of these complications we cannot control the lower fragment by the ordinary conservative method of treatment. At this stage, seven weeks after the accident, we must attempt to reduce the deformity and to apply some type of fixation to the fragments. The type of fixation that is suitable for this case is the nailing type—the extra-articular bloodless operation. In a Colles's fracture you do not have to retain the lower fragment by nailing, except in very rare instances (see Clinics, August, 1914, pp. 731–743), because after complete

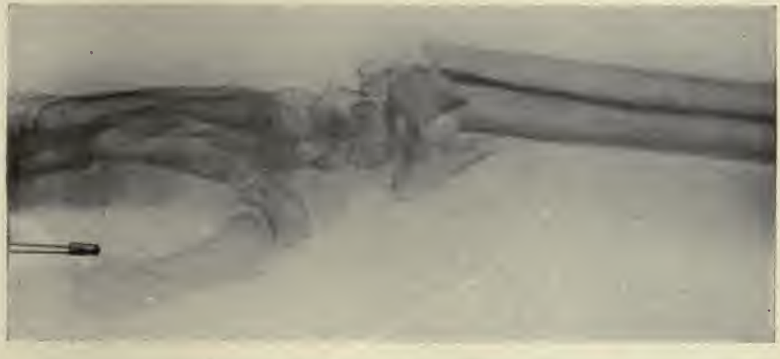

Fig. 295.—Preoperative skiagram of hyperflexion fracture involving distal portions of radius and ulna. Note these features: (1) Oblique plane of fracture which runs from behind and below, forward and upward; (2) volar displacement of lower fragments bearing the carpus and the hand; (3) dorsal projection of sharp edges of lower ends of upper fragments; (4) the manner in which the extensor tendons are exposed to trauma from the sharp bony edges (lateral view).

reduction of a Colles's fracture the lower fragment will remain in place even if you dress the fracture merely in a sling. The great mistake, and the cause of future trouble in the management of Colles's fractures, is the effort made by the surgeon to reduce the fracture by the mere application of a splint. *Splints were never intended to be used for the purpose of effecting the reduction of fractures.* They were designed to serve as an agent merely for immobilizing the fragments *after* the reduction has been accomplished.

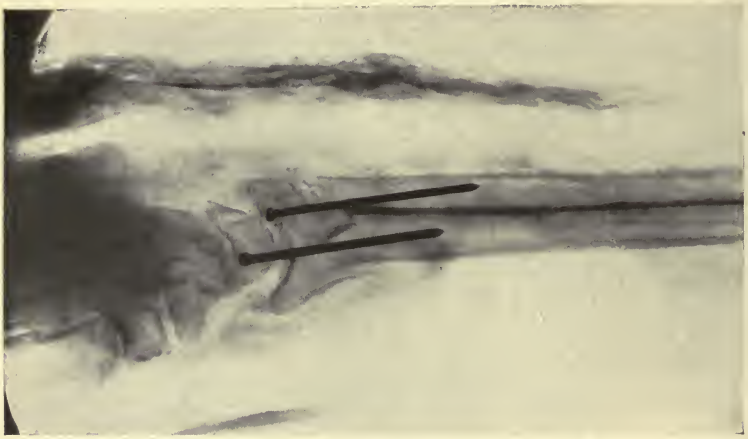

Fig. 296.—Postoperative skiagram showing reduction of fragments and maintenance of reduction by extra-articular fixation with ten-penny "finishing" nails. Compare with Fig. 295. The nail in the radius was driven through the outer comminuted fragment (*cf.* Fig. 292) (lateral view taken through plaster-of-Paris dressing).

OPERATION

[Manipulates seat of fracture.] We are loosening up these fragments in an endeavor to restore them to their normal relationship with each other . . . The deformity is now fairly well reduced (Figs. 294, 296). Next we shall nick the skin over both styloid processes as the first step in the operation of extra-articular bloodless nailing. There is one landmark—the styloid process of the ulna. [Nicks skin over the styloid process.] Now I am inserting a single ten-penny "finishing" nail on this, the ulnar side (Figs. 294, 296). . . . There is the styloid process of the radius. [Nicks skin over the styloid process.] Note that, when nicking the skin at this point, I am not compounding the fracture, because I do not open to the line of fracture at all. The lower fragment of the radius is split, and we want to keep to the inner side of the plane of splitting if we can . . . I am inserting a single nail on the radial side . . . Now you can understand what is meant by the operation of extra-articular bloodless fixation, since you saw me drive the nail through both fragments without entering the joint. When you look at that hand you see that it is perfectly straight (Figs. 294, 296). All that remains to be done is to dress the forearm upon a straight piece of wire gauze or a straight molded splint of plaster-of-Paris.

POSTOPERATIVE COMMENTS

Now let us take up the proposition of the reduction of the most frequently maltreated fracture that occurs in the body, and the one that, unreduced, gives the most evil result—a Colles's fracture. The plane of this fracture is always oblique from below and in front upward and backward. The degree of displacement of the lower fragment varies in different cases. Sometimes the displacement amounts to just a slight angulation through impaction of the lower end of the radius, while at other times the lower fragment is displaced away up on the dorsum of the upper fragment. Is that clear?

The nature of this deformity is the first thing you must think of when dealing with a case of Colles's fracture. With the upward and backward tilting of the lower fragment the angle of the carpal articular surface of the radius will be altered, so that, in place of its facing normally slightly forward, to facilitate flexion of the wrist, it faces backward. It is this backward displacement of the lower fragment that gives rise to the fork-handle deformity. Such a deformity was not present here at all. This picture illustrates the deformity beautifully. [Demonstrates a skiagram of a Colles's fracture.] The lower fragment, you see, is displaced upward and backward. The articular surface, in place of facing slightly forward, is inclined backward, and this inclination must needs carry the carpal bones backward, altering the mechanics of the wrist-joint and exposing the joint in its movements to traumatic irritation. In addition to the fork-deformity shown in this skiagram, there is separation of the ligamentous and cartilaginous attachments of the lower end of the ulna to the carpus. Here is an illustration again of the operation of extra-articular bloodless fixation by nailing. We had to insert that nail because the lower fragment of the radius did not remain in position. The patient came in late after the injury, so that we had to operate, free the callus, and swing the displaced lower fragment around and nail it in position.

Now let me show you the proper method of reduction. [Employs a manikin.] We'll imagine that this man has just come in with a Colles's fracture. Picture the deformity—the lower fragment displaced upward and backward and overlapping the upper fragment. [Illustrates extent of overlapping.] Now, then, if in your attempt at reduction you merely pull upon the hand in the long axis of the forearm, you may succeed in bringing the two fragments parallel with each other, but not parallel in the same plane. Merely taking the hand and pulling upon it does not unlock the serrated and usually impacted edges of the fragments: you may succeed in altering the angle, but not in reducing the deformity. Here the principle is wrong, and the result a failure.

What is the correct method of effecting complete reduction? The only way by which the fracture can be reduced properly is by increasing the dorsal deformity almost to a right angle, unlock the fragments, push the lower fragment downward and flex it forward, at the same time swinging the hand well around to the ulnar side. No matter how you dress the forearm, with the final shifting of the displaced fragment back into place the work is completed. In a Colles's fracture the crux of the treatment is purely and entirely a matter of reduction. *The reduction cannot be accomplished unless you first unlock the fragments.* No matter how much you pull on the wrist, nor how much you force it forward, you cannot effect reduction unless you unlock the fragments first.

How do you accomplish this unlocking? Always with the patient anesthetized. Place the thumb of the distal hand upon the posterior surface of the distal fragment and the thumb of the proximal hand upon the nail of the distal thumb, and then increase the deformity thus. [Demonstrates on manikin the method of increasing the deformity by hyperextension.] With the great degree of pressure used I could break this normal radius with ease. If, when the thumbs are removed, the contour of the forearm at the site of fracture does not look perfectly smooth and flat, the lower fragment has not been reduced; and if, at the time of the next visit, the patient tells you that he was kept awake all night by the pain, the fracture is still unreduced. *A properly reduced Colles's fracture is painless.* With the reduction finally accomplished, dress the forearm by a few recurrent turns of a plaster-of-Paris bandage placed upon the posterior surface so as to cover three-fifths of the circumference of the forearm.

One of the great annoyances sequential to improper reduction of a fracture of the Colles's type is that of volar projection of the lower end of the upper fragment, and in such a manner that the flexor tendons are held bound in between the lower end of the upper fragment of the radius and the annular ligament, causing the patient a great deal of pain. Patients with an unreduced Colles's fracture will come back to you not because of the deformity, but because of the inefficiency of the lamed hand—lamed by the tightly held tendons riding over the edge of the upper fragment. Should it fall to your lot to treat an ancient, unreduced Colles's fracture, you must perform osteotomy and make a V-shaped division of the radius at the site of fracture. Split the bone in this direction [indicating], so that you cut it in the shape of a double "A." Then, after cutting clear through the plane of fracture, you can adjust the lower fragment in any direction you wish, according to indications. The reduced fragment is to be retained in this favorable position, if there is still a tendency to displacement, by inserting a single ten-penny nail, which easily suffices to hold it. Such a later operation is a secondary operation: a primary operation for a Colles's fracture is rarely required.

This patient injured the opposite (right) hand last winter (January, 1915). She suffered an impacted Colles's fracture, which shows clearly in the skiagram and just as clearly upon inspection. There was also a fracture of the os magnum, which is a very rare fracture. The Colles's fracture was untreated because unrecognized until after union had occurred. This unreduced fracture accounts for the thickening which you see in the hand and wrist, and this thickening has very materially impaired the use of the hand. In an attempt to overcome some of the evil effects of this ancient unreduced fracture with vicious union we are loosening up these bound-down tendons.

[In addition to the previously published case of Colles's fracture cited above in the text, another case was presented in the Clinics for June, 1912, pp. 315–323.]

EXTENSOR CONTRACTURE OF HANDS FOLLOWING BURNS—EXCISION OF CICATRICIAL TISSUE—GRAFTING BY PEDICLED ABDOMINAL FLAP

Summary: Clinical history of patient; comments—discussion of deformity; danger associated with annular scars and with scars in flexures of joints; plan of operation for permanent relief of contracture; operation—removal of scar-tissue from dorsum of hand; elongation of extensor tendon of thumb; preparation of abdominal flap; suturing of flap to dorsum of hand; dressing and after-treatment; postoperative comments—necessity of removing whole area of cicatricial tissue preparatory to grafting; great vitality of pulp of finger-tips; postoperative release of graft by dividing pedicle; citation of similar case, but one in which flap from abdominal wall shares in general obesity.

CLINICAL HISTORY OF PATIENT

The patient, a male aged twenty-four years, was admitted to hospital May 30, 1915. The family and past histories are negative.

Nine months ago, on August 22, 1914, while driving an automobile through an oil district, the gasoline in the tank of his car exploded, and the patient sustained severe burns of both upper extremities, of the face and ears, and of a large area of the right hip. He has had repeated graft-operations performed upon these areas. The burned areas have practically all healed, with the exception of the ones on his hands. There is very little motion in some of the fingers. The right arm cannot be extended beyond a right angle.

Examination shows that the patient is able to flex the fingers of the left hand as far as the contracted cicatricial tissue on the dorsum will permit. There is an annular contracture on the forearm.

COMMENTS

Dr. Murphy (June 2, 1915): The patient comes to us to see if we can restore the power of closing his hands. His fingers remain hyperextended (Fig. 297, *A*), and he is unable to close the hand. There is no grip at all in either hand, because of so much scar-tissue on the dorsal surface. The palmar surface is smooth, and is not involved by enough scar-tissue to interfere with movements, but the contracture on the dorsal surface is so great that he is unable to use his hands for any purpose whatsoever.

There is still another proposition to consider. The patient has on his forearm a large, annular burn-scar, which involves the entire circumference of the forearm. Wherever an annular scar involves the entire circumference of an extremity, there is always danger of the cicatricial contraction interfering with the circulation of the hand or foot below, and finally destroying it. An annular scar is always a source of danger, particularly when located in any of three positions.

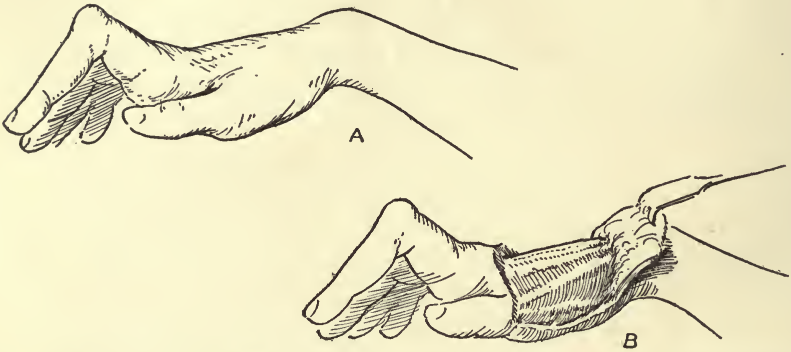

Fig. 297.—*A*, Clawhand from contracting scar on dorsum. Note hyperextension of first phalanges and flexion of the second and third. *B*, Cicatricial area of skin removed, preparing bed for pedicled skin-graft.

Many years ago there was in vogue an operation for varicose veins of the leg, known as the Schede operation, which resulted in the establishment of a complete annular scar just below the knee. At one time we had in the Cook County Hospital two cases of gangrene of the foot—both the sequence of contraction of the completely annular scar that developed after the Schede operation. Again, after operating for hemorrhoids, where an annular incision into the skin has been made, a keloid not infrequently develops, and by its contraction finally leads to stenosis and occlusion of the anus. The third position where scars cause mischief is in the flexure of the elbow, of the popliteal space, the palmar surface of the hand, and the anterior surface of the wrist. In all operations upon these flexure-sites you should endeavor to avoid making a long incision parallel with the long axis of the extremity across the transverse crease of the elbow, or of the popliteal space, since in both these flexures the expansion and contraction of the connective tissue of the scar stimulate the excessive formation of fibrous tissue and lead ultimately to the development of a keloid.

There is the annular scar on the forearm—it is completely annular. The forearm is flexed more and more by the contracture, and continuance of the flexion may lead to stagnation of the circulation in the forearm below, unless some step be taken to release that long, annular scar. From the patient's standpoint, this is a beastly situation; and I may add that, from the surgeon's standpoint, it is anything but attractive. At the same time it is not fair to this patient to say that nothing can be done, or that no attempt to relieve him should be made. Both his hands will remain permanently out of commission unless we should do something for him.

The scheme we have outlined for his relief is to transplant, onto the dorsum of the hand, opposite the knuckles, a strip of normal skin that is broad enough to fill in the gap left after excision of the scar-tissue, as well as release the knuckles and permit the fingers to come forward. If we can excise the whole area of cicatricial tissue on the back of the hand and insert into the denuded area a flap of normally elastic skin,—say from the abdomen,—that flap will stretch from 30 to 40 per cent. of the size it was when inserted, and will finally permit the fingers to be flexed and to be of service to him. Furthermore, there will be no stimulation of connective-tissue formation when elastic, normal tissue is interposed between the remaining ends of the interrupted annular cicatricial tissue: his fingers cannot be flexed until this plan has been executed: no amount of pressure that I am able to exert upon the fingers will bring them farther forward than they are, so firm and unyielding is the scar on the dorsum.

OPERATION

[Incision.] I am going through the cicatricial tissue and freeing all the scar-tissue and laying it back out of the field, so as to let it retract far above and far below. I cannot transplant these tendons at present: if I should transplant them now, the flap would have a raw surface under it, and there would be some danger of infection of the raw surface. I am elongating the extensor tendon of the thumb so that I can bring that digit around in a little better position for leverage action than it is at present. We should like to do this with all the tendons, but should this wound suppurate, as it could and might, then we would have destruction of the power of extension. . . . Now we are getting this flap freed upward as much as we feel is necessary for the transplantation. . . . You see that we have finally prepared the bed for the transplant (Fig. 297, *B*).

Now I am preparing to elevate the flap from the abdomen. In such a flap you must include all the fatty tissue you are able to obtain from the operative site. If you do not include a great deal of fat, the flap will be a failure. [Makes parallel vertical incisions between left nipple and umbilical zones (Fig. 298, *C*).] See! I am cutting clear down to the sheath of the rectus muscle. Note that I am including in the flap the entire thickness of the skin and superficial fat of the abdominal wall . . . The next step is to suture the far edges so as to restore the continuity of the skin of the abdomen (Fig. 298, *D*). I am approximating these edges in such a manner that each new stitch takes the tension off of the previous stitch, and those in the middle will be free of tension when the upper and lower stitches are tied. I have now elevated the transplant and accurately apposed the tissues beneath it, in my endeavor to secure union of the skin-edges left remaining after elevation of the flap (Fig. 298, *E*). There is now no undue tension upon the line of sutures.

The flap we hold in the retractor is of the proper size and shape to bridge over the denuded area on the dorsum of the hand (Fig. 298, *E*). [Passes patient's hand beneath flap with palm facing abdomen and dorsum in contact with under surface of flap, and then sutures skin-edges of hand-wound to skin-edges

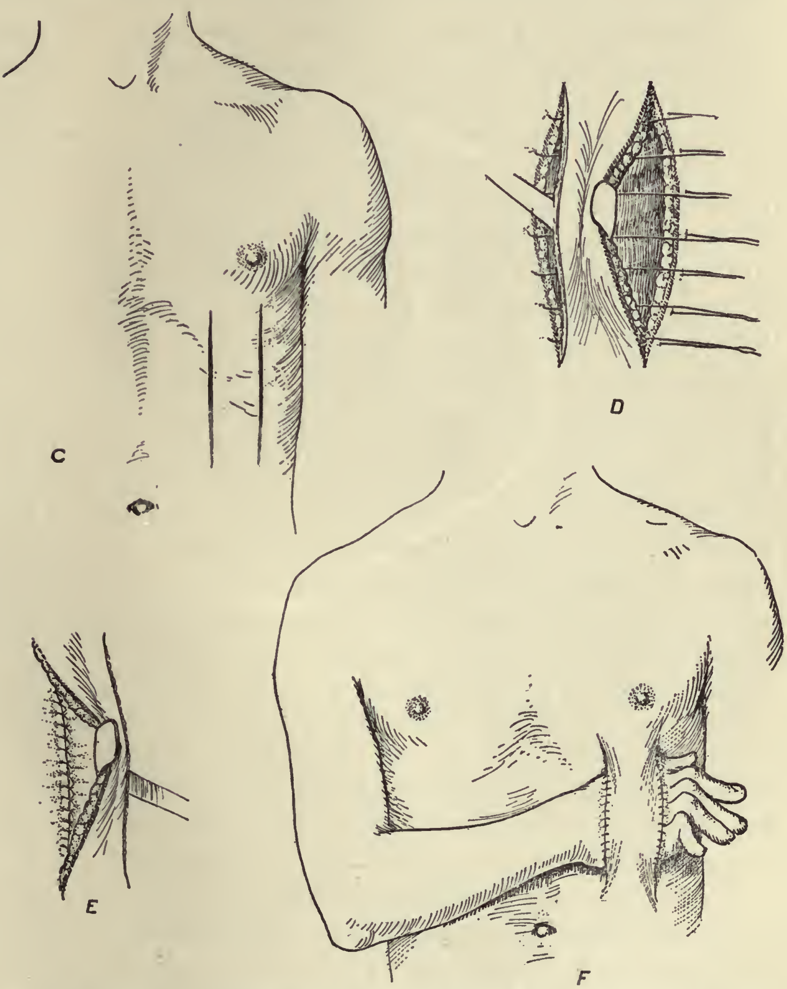

Fig. 298.—*C*, Development of pedicled skin-graft. Two parallel incisions made upon front of left chest below nipple; *D*, skin-graft freed except above and below and elevated by retractor. Insertion of approximating sutures for closure of wound behind graft; *E*, approximating sutures tied, contacting edges of wound beneath elevated pedicled graft; *F*, hand "strapped" to chest by skin-graft, which lies in its bed sutured to the skin-edges of the dorsum of the hand.

of flap (Fig. 298, *F*).] Now, you see, I have, figuratively speaking, put a mitten on the back of this hand, and in raising the flap I included all the fat and fascia that was available in the region, so that the flap can stretch extensively when we begin postoperative manipulations upon the hand. At first glance it looks as though there were a great surplus of tissue here, but, on the contrary, when we get through, we shall probably be short of tissue, even with all the flap that you see here spanning the knuckles. The edges of the flap are contacted with edges of normal, healthy skin on the dorsum of the hand.

Dressing and After-treatment.—This hand will have to be fixed to the patient's body in perfect immobilization for from two and one-half to three weeks, the time depending upon how rapidly the circulation in the transplant develops. If the circulation develops rapidly and there results firm union without suppuration at the angles, where the wound is more or less exposed to infection, we shall cut the pedicles of the graft before that time. [Applies adhesive plaster to retain arm in contact with abdomen.] This adhesive plaster does not obstruct the circulation of the hand. In immobilizing the arm we want to make it as comfortable as possible, for it will be annoying as it is, and the patient will be greatly distressed before the time for release from the trying position has arrived. The arm will be kept perfectly still until we feel that the graft has become firmly united to the skin of the hand. You should cover the dressing with cotton, like you cork a culture-tube, packing plain, sterile cotton around so as to prevent the microörganisms from getting beneath the dressing and in the wound.

POSTOPERATIVE COMMENTS

In 1886 we called attention to the fact that, when one performs a grafting or transplantation operation, in the preparation of the ulcer field, he must go clear down to normal tissue; that is, he must get beneath the entire depth of the cicatricial tissue. Otherwise, when the patient gets about and begins to use the limb, the connective tissue that is left remaining beneath the flap, or beneath the graft, begins to contract, and, contracting, strangulates the blood-vessels that supply the graft, with the result that the graft necrotizes. I recall a case of this kind in which there was an extensive burn of the leg. I made a homograft from the tips of the fingers of an amputated hand of a patient who had come into the Cook County Hospital at the same time, and applied them to the scar-denuded area of the leg. The grafts grew beautifully—every graft lived. Remember, the tips of the fingers on the palmar side have a greater degree of vitality than any other tissue in the body except the tip of the nose. After we let this patient get up, and as he began to get about, the grafted area became gradually more and more edematous, and soon the grafts began to die in the center, until finally all the grafts ulcerated and were discharged as sloughs. Later, when we reoperated, we found beneath the grafted area a mass of contracted cicatricial tissue that we had not excised. From this case we learned a lesson, and since that time have paid particular attention to the removal of the whole area of cicatricial tissue in order to secure an ample blood-supply through the base of the flap.

Release of Graft from its Pedicles.—DR. MURPHY (June 23, 1915—three weeks after operation): Upon removing the dressing we find that this whole graft has survived. Aided by manipulations, it will expand from 40 to 60 per cent. of its original width. We anticipate much improvement in the position of the patient's fingers. We intend performing a similar operation upon the other hand later.

The patient is to return in about eight months, when we shall probably elongate all the extensor tendons, because they have contracted so much; and until then it will be impossible for the patient to have much function in these fingers.

[*N. B.*—In the *Hygiea*, Stockholm (lxxvii, No. 7, pp. 353–400—abstract in *Jour. A. M. A.*, June 19, 1915, p. 2106), in an article entitled "Flap from Abdominal Wall Shares in General Obesity," Strandberg reports the case of a girl of twelve who had a defect in the back of her hand remedied by a flap taken from her abdomen. It healed in place and answered its purpose perfectly until late in life. About eighteen years later, after the age of thirty, the patient became obese. Then the grafted patch on her hand increased in size proportional to the increasing thickness of the abdominal wall (Fig. 299).]

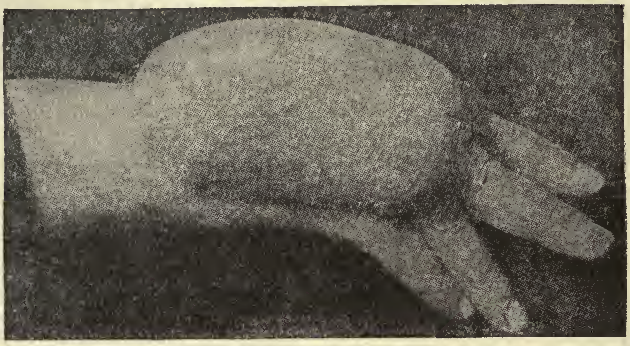

Fig. 299.—Flap taken from abdominal wall shares in obesity (Strandberg).

[An operation somewhat similar to the above, but upon the fingers, was published in the Clinics for February, 1915, pp. 155–164.]

OSTEITIS FIBROSA CYSTICA OF PHALANX OF FINGER—CURETTAGE OF CYST CAVITY AND INSERTION OF TRANSPLANT FROM TIBIA

Summary: Clinical history of patient; comments—varieties of bone-cysts and of bone-tumors; metastatic thyroid tumors—sites commonly involved by bone-cysts; diagnosis and treatment of *osteitis fibrosa cystica;* operation—curettage of cyst and insertion of transplant from tibia; recapitulation; references to other cases of osteitis fibrosa cystica.

CLINICAL HISTORY OF PATIENT

THE patient, a male aged forty-five years, a physician, was admitted to hospital May 11, 1915. The family and past histories are negative.

Over three years previously, in December, 1912, while engaged in washing his hands, the patient felt a sensation of soreness in the proximal phalanx of the middle finger of the left hand. On examination, he found that phalanx tender to pressure and felt a little elevation on the side next the ring finger (Fig. 300, 1); he then recalled that several times in the recent past the ring on the ring finger had pressed against the involved portion of the middle finger. A physician advised amputation of the finger, but to this the patient did not consent. He took no further action in the matter, but was careful not to strike the lesion, nor make pressure upon it, since the finger ached when traumatized. There were occasionally sharp, shooting pains in the digit.

Three days previous to admission, on May 8, 1915, while lifting a door, the patient struck the side of the door with his left hand, whereupon he immediately felt the finger crack, and on examination found that it was broken and deformed. Since the receipt of the injury there has been constant severe pain in the finger. His general health is good.

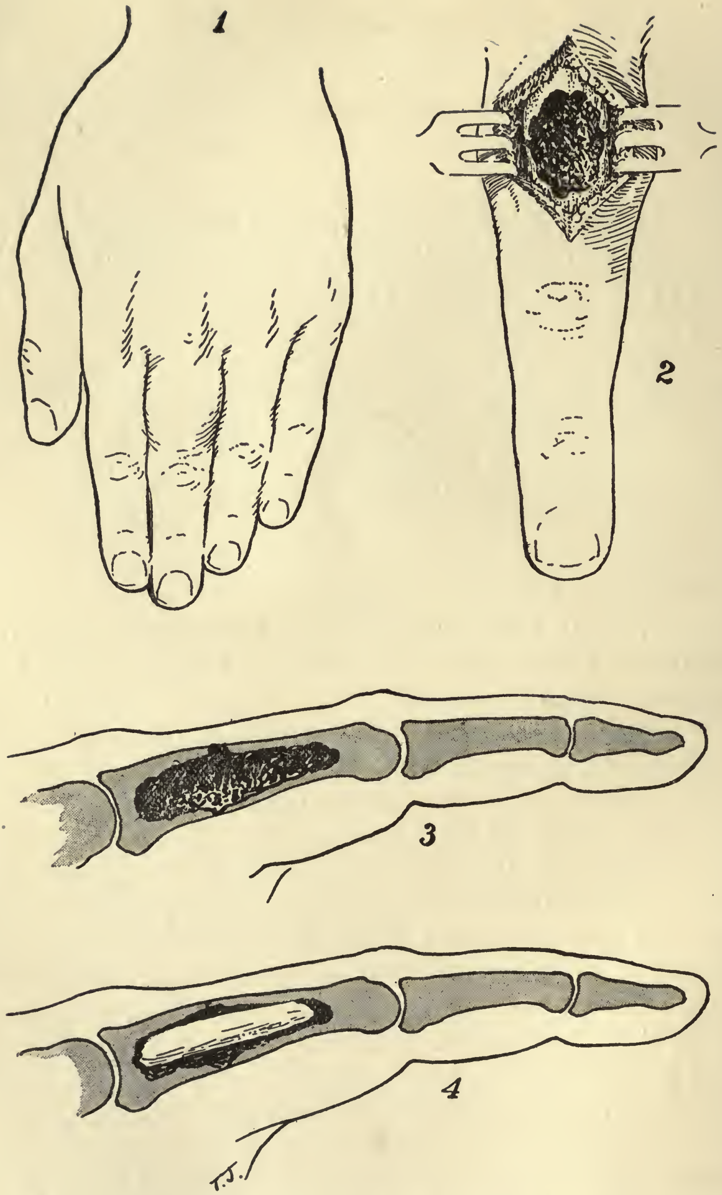

Fig. 300.—Series of sketches illustrating case of *osteitis fibrosa cystica* of phalanx of finger, and operative measures for its cure. *1*, Shows enlargement of proximal phalanx of middle finger, more marked at ulnar border. *2*, Longitudinal incision made on dorsum of cyst; retraction of wound-edges; removal of wall of cyst, revealing gelatinous contents. Pathologic fracture shows at middle of broken edge of cyst wall. *3*, Schematic longitudinal section of finger showing extent of cyst in proximal phalanx; note pathologic fracture. *4*, Same as *3*, showing transplant from tibia ***in situ***.

COMMENTS

Dr. Murphy (May 12, 1915): What is the matter with the patient?

Intern: I think that there is a pathologic fracture of the finger ——

Dr. Murphy: Correct. Go on.

Intern: —— and that it was brought about by the disease in the bone. You have to consider two lesions—sarcoma and bone cyst.

Dr. Murphy: What varieties of essential bone cysts are there?

Intern: One variety is *osteitis fibrosa cystica.*

Dr. Murphy: What other types of cysts do you have?

Intern: Hemorrhagic cysts.

Dr. Murphy: That variety is now believed to be one of the rare types due to attenuated infection. You have tumors of the compact bony tissue. You have bone tumors resembling marrow tissue,—the myelomata, or giant-cell sarcomata,—which are commonest in young subjects and most frequently found at the lower end of the femur, the upper end of the tibia and fibula, and in the mandible. Then you come down to the primary and secondary tumors of the malignant type. Of the malignant tumors, carcinoma is always secondary and never primary. You have next the sarcomata, which occur primarily or secondarily, and more commonly the latter than the former. Then you have the various subdivisions of the sarcomata, which differ according to the type of cell- and alveolus-formation. Then you come to the thyroid tumors. What do you mean by a thyroid tumor?

Intern: A thyroid tumor of the bone is a metastatic lesion.

Dr. Murphy: What do you mean by metastatic thyroid tumors?

Intern: Innocent or malignant tumors.

Dr. Murphy: What are they, and where do they occur?

Intern: They occur in bone.

Dr. Murphy: The innocent thyroid tumors occur most commonly on the calvarium: they also involve the flat bones elsewhere in the body,—the ilium, the scapula, and occasionally the bodies of the vertebræ,—but the commonest place is the calvarium. Upon operative exposure of the tumor you find, both macroscopically and microscopically, the structure of a normal thyroid gland.

The other variety is the malignant type of thyroid tumor in bone, and occurs as a metastatic growth from the thyroid gland. It involves the shaft of long bones, usually the metaphysis, rarely the epiphysis. We know that there is a direct relationship, physiologically, between the thyroid gland and the shaft of long bones, because the growth of the shaft of long bones depends upon the functional activity of the thyroid gland and the presence of its internal secretion in the body. If you excise the thyroid gland, the long bones cease to grow, and this is illustrated by the cretin, who does not grow because of his deficiency in thyroid secretion. Therefore, when a thyroid gland is carcinomatous, metastases not uncommonly travel to a bone, but *not* most commonly to a bone, but to the lungs and pleuræ, since from all carcinomata that are situated above the clavicular girdle metastases most commonly travel to the lungs and pleuræ.

This cyst is of the osteitis fibrosa type. Such cysts occur in various localities in the body, and most commonly in the upper end of the humerus; next in frequency in the upper end of the tibia; then in the phalanges, and in the upper end of the femur. I am showing you a specimen from a case of *osteitis fibrosa cystica* that began in the upper end of the humerus: note how the cyst spread through the shaft and on down the bone until more than 50 per cent. of the shaft of the humerus was involved.

You can make the diagnosis in these cases from the skiagram by the fact that a shell of bone is not elevated in an irregular manner by the cyst, as obtains in sarcoma. Here is one of our early (1910) cases of osteitis fibrosa cystica of the upper end of the humerus: we removed all the involved bone and transplanted a bone-graft 7 or 8 inches long from the tibia into the humerus. These skiagrams show how completely the contour of the humerus has been restored by reproduction of bone along and around the transplant. The patient holds her arm out perfectly straight. She is quite well, and would not know that anything had ever been done to her arm.

Here is another cyst, involving a large extent of the humerus. Here is a cyst involving the femur: in operating we curetted the cyst and then inserted a transplant. Here is Dr. Golden's case of a cyst involving a considerable extent of the tibia. The diagnosis is clear: seeing these cystic areas, you can tell at once that we are not dealing with a sarcoma, but with *osteitis fibrosa cystica*, since the shell of bone has not been elevated in an irregular manner.

There is the area of thickening (Fig. 300, *1*), and here, the point of pathologic fracture. Owing to the small size of the phalanx, we cannot use the inlay method as we did yesterday, when operating upon the case of fractured ulna, and yet the finger will not have to be amputated. You can remove with the curet the entire contents of the cyst until only a shell of bone remains, and then insert a transplant, which lessens the liability of a return of the disease and prevents the deformity of the finger during bone-regeneration. The process of repair is accelerated by leaving the periosteum on the transplant.

OPERATION

I do not wish to open into either of the adjacent finger-joints in exposing the cyst, but intend going beneath the extensor tendon and elevating the periosteum. [Longitudinal incision made on dorsum over tumor; tendons and subcutaneous tissues retracted (Fig. 300, *2*).] Now you see exposed here the expansion of the bone at the site of the tumor; there is the line of pathologic fracture. [Enters cavity of cyst by breaking through its shell.] There is the granulation-tissue within the cyst-cavity. I wish to have this specimen preserved for the microscopist.

I shall take out a quantity of this granulation-tissue and show it to you: it is not sarcomatous. Had the patient not been a physician, he would have had this finger treated before it was pathologically fractured. I shall request the pathologist to make cultures from the granulation-material in this cyst and keep them in the incubator for twenty days. Remember, the etiologic factor in Hodgkin's disease was overlooked because the cultures were not kept growing long enough to reveal the diphtheroid bacilli; it takes eleven days for the bacteria in the culture to begin appreciably to multiply.

Had this cyst been treated early in the course of its development, it would not have been necessary to transplant bone. I shall remove a piece of bone of suitable size from his tibia with a chisel, because I can do it quicker with a chisel than with a saw. In removing a graft from the tibia the skin is incised a little to the outer side of the crest of the tibia, and the tissues are retracted. I am loosening the periosteum and peeling it down.

RECAPITULATION

Let the record show that on exposing the phalanx we found the point of pathologic fracture; that on opening the tumor it was found to contain the typical gelatinous material; that we removed with the curette the entire contents of the cyst; that we then obtained a transplant from the tibia and inserted it into the cavity of the cyst (Fig. 300, *4*); and that during the operation there was practically no bleeding, the field remaining perfectly dry, which is typical for these cases.

[*N. B.*—There was primary union and good preservation of the conformation of the finger.]

[The cases of *osteitis fibrosa cystica* referred to above have been published in the Clinics as follows: of tibia, August, 1912, pp. 555–569; of humerus, October, 1912, pp. 741–747; of femur, October, 1913, pp. 783–805. An abstract of cases of cyst of the metacarpal bones has been presented by Dr. Murphy in the General Surgery Volume of the *Practical Medicine Series*, 1915, p. 542.]

MULTIPLE ANGIOMATA—ABLATION BY KNIFE

Summary: Clinical history of patient; comments—a cyst anywhere in body is most painful when small; the term "neurasthenia" a meaningless term; two ways in which angiomata may terminate life; danger of this neoplasm and similar neoplasms becoming malignant if traumatized; angiomata apparently intradermal, but in reality subdermal and therefore more liable to undergo malignant change; *adiposis dolorosa*—symptoms, sites, specific cure; operation.

CLINICAL HISTORY OF PATIENT

The patient, a female aged twenty years, was admitted to hospital June 1, 1915. The family and past histories are negative.

The first angioma appeared as a small red spot on the patient's back when she was about three months of age. This angioma slowly increased in size, and one year later similar spots were noticed in various parts of the body. When the patient was three years of age one of these angiomata was removed from the inside of her cheek; and ten years later, in November, 1908, two were removed from the face and one from the subpatellar bursa by Dr. Murphy. In no case was there local recurrence after excision. Several angiomata have appeared in the palms of both hands and in the soles of both feet, but these have grown very slowly. The appearance of each new angioma is accompanied by definite pain, which lasts until the angioma has reached a considerable size, whereupon the pain disappears. The patient's mother pricked several angiomata with a pin and evacuated their contents, but all refilled within the course of a few weeks.

Four months previous to admission the right breast began to be painful, and shortly afterward the left breast followed suit. The pain, which is sharp and aching in character, is aggravated by the menses. The breasts have not become enlarged. Four years ago, in 1911, an angioma appeared in the conjunctiva of the right eye and has slowly increased in size.

COMMENTS

Dr. Murphy (June 3, 1915): That story is very typical and is heard from patients with an ovarian cyst or a cyst of the kidney: in fact, it is heard from patients with a cyst anywhere in the body. The story is that the cysts are most painful when small. A woman who has an ovarian cyst as large as one's head suffers no pain at all; but during the time of its evolution she did suffer most when the cyst was as small as a walnut—while the tunica albuginea of the ovary was being stretched. It is in this stretching stage that patients with small ovarian cysts come to us, and as we find nothing upon examination, we conclude that they are neurasthenic, which term, when properly interpreted, means nothing. The term "neurasthenia" has the same empty value in diagnosis as the term "rheumatism," but it is a little more complicated in nomenclature—a little harder to spell or pronounce. Such loose terms really signify that you do not know: that you have not found out.

It was dangerous for the patient's mother to prick these tumors and evacuate their contents. The angioma which I removed seven years ago from the subpatellar bursa presented an extensive bulging beneath the ligamentum patellæ, forcing the fatty capsule into the joint. I thought that the angioma involved the joint, and that we would have the annoyance of its developing to a great size in the joint, but, fortunately, it did not: it was cured by the operation and has remained well since. At that time none of these angiomata was present that she has now.

We first saw this patient seven years ago, in 1908, when she was thirteen years of age. Since the original operation at that time the angiomata have been increasing in number.

What will be the further course of these angiomata? One of two terminal events may occur: either an angioma may develop in the meningeal veins or in those of the choroid plexus in one of the ventricles, causing rise of cerebral pressure from the angioma growing large, giving symptoms of ataxia, atrophy of the optic nerve, and finally causing death; or else one of the angiomata may be traumatized, become malignant, and eventually cause the death of the patient from metastases to vital organs. By way of illustration, take pigmented moles anywhere in the body: when traumatized, they often rapidly undergo the most wicked type of malignant changes. I have frequently cited in this clinic the case of a young woman who had one of these little pigmented moles in the skin over the spine of the scapula. She was departing from an afternoon tea one day, and a lady said to her, "That spot on your back looks so much like a fly that I cannot refrain from swatting it," and she *did* swat it. Slowly, but surely, the mole began to enlarge. It was removed by operation, but, owing to recurrence, we were compelled to excise the entire scapula. Three months after this operation the neoplasm showed metastases in the lungs, and the metastases caused the patient's death. There was no local recurrence in the shoulder. Such observations impress upon one what potential dangers lurk in these neoplasms, even though they be, at first glance, such small, insignificant affairs. This patient's angiomata have been traumatized frequently, but have not yet become malignant.

There is an angioma in her axillary fold that is annoying her. Here is another in the conjunctiva, which looks ugly to me: it has a peculiar feel, but I do not think that it is malignant. There is a third angioma on the buttock—in a position to be traumatized: that is one of the reasons for removing it. You note that, while all these angiomata are apparently in the derma, yet they are not intradermal, but *subdermal*—in a site that is dangerous insofar as malignancy is concerned.

This patient is a very large woman for her age. Within her breast is a tumor which is either an angioma or a lipoma, I do not know which. It may be a painful accumulation of fat and due to *adiposis dolorosa* (Dercum's disease), which is not at all common in the presence of an enlargement of the thyroid gland, such as hers. Adiposis dolorosa is usually associated with *deficiency* of the thyroid secretion or hypothyroidism. Adiposis dolorosa, which means painful subcutaneous fatty masses, involves more commonly the external surface of the thigh; next the internal surface of the upper arm; then the abdomen, and finally the chest and the breast. These accumulations of fatty tissue are extremely painful—both upon pressure and without pressure—and greatly annoying to the patient. Sufferers resort to anodynes and usually become addicted to the use of those drugs from which they have obtained relief.

If one should recognize adiposis dolorosa as the lesion,—and one *can* recognize it if he be alert,—he has at hand, in desiccated thyroids, a practically specific method of treatment. Why this agent should cure no one has yet explained. Cure is brought about by the daily administration of desiccated thyroid gland in doses large enough to intoxicate the patient—the intoxication being manifested by such signs as tremor and tachycardia, typical of Graves's disease. These signs appear after patients have been taking six, seven, eight, nine, or ten grains of desiccated thyroid gland three times a day after meals. Such patients tell you that they are trembling all over, and that they feel as though they were going to shake to pieces. When these signs of thyrotoxicosis appear, you will know that you are obtaining the full therapeutic effects from the medication, and that you will soon be getting definite results, as shown by the disappearance of the adiposis dolorosa under the administration of the desiccated thyroid gland.

OPERATION

In making the incision I am going wide of the tumor. . . . In ablating the tumor one should never traumatize it by grasping it with the forceps. Such a trauma of the tumor may be avoided by grasping with the forceps the skin margin left remaining between the periphery of the tumor and the incision, which was made wide of the tumor. In this manner the latter may be elevated and then separated from the underlying tissues by dissecting well beneath the base of the tumor. [Wound left remaining after ablation of angioma closed in usual manner.]

[A case of angioma involving the lower extremity was published in the Clinics for August, 1912, pp. 497–508; a case of angioma of the thigh was published in the Clinics for October, 1913, pp. 807–809.]

BILIARY CALCULUS IMPACTED AT AMPULLA OF VATER—MOBILIZATION OF DUODENUM—DUODENOCHOLEDOCHOTOMY (McBURNEY)—EXTRACTION OF CALCULUS

Summary: Clinical history of patient; examination; comments—analysis of symptoms; composition of biliary calculi; operation—adhesions dissevered; calculus felt, impacted in ampulla of Vater—extraction by transduodenal route; recapitulation; postoperative comments—postoperative treatment; correlation of symptoms and signs with operative findings; review of steps of operation.

CLINICAL HISTORY OF PATIENT

The patient, a male aged fifty-nine years, was admitted to hospital August 31, 1915. The family and past histories are negative.

For the twenty-five years previous to 1914—from 1889 to 1914—the patient complained of sharp, cutting pains in the epigastrium, which pains radiated straight backward to the vertebral column at the same level. At first the pains were not severe and occurred every two or three months only. There being no symptoms other than the pain, the malady was treated as gastralgia and dyspepsia.

By January 15, 1911, the pains had become very severe, lasting an hour, and returning within a week, always appearing in exactly the same place, and always radiating from just beneath the ensiform straight through to the back. Two weeks after the colicky pains became severe, on February 1, 1911, jaundice appeared for the first time, lasted three weeks, and then cleared up. The severity of the pain was mitigated by the administration of purgatives. There were no pains elsewhere, nor chills or sweats; there was no nausea, vomiting, or fever, and the stools were normal in color and consistence. For the next three years the patient had attacks of pain only occasionally.

On January 5, 1914, severe pains, followed by jaundice, reappeared and lasted six weeks. Following the onset of the sharp pain in the epigastrium, which lasted less than two hours, the whole abdomen ached. There were still no chills or sweats, nor was there any nausea, vomiting, or fever. The stools and the urine were normal.

One month later, in February, 1914, while the patient was still jaundiced, he was operated upon, and the gall-bladder was found infected and contained inspissated bile. Cholecystectomy was performed. A gauze drain was inserted into the operative field. For two days after operation the patient vomited blood; the jaundice disappeared within three days. The wound drained bile for seven weeks, when the sinus closed, and there has been no sign of discharge since. The jaundice appeared immediately after the cessation of drainage, the stools becoming light yellow and semisolid, and the urine reddish-green in color. For the past three months the stools have been brick-red in color, while the urine has been tinted a dirty, reddish-green. There have been no other symptoms, except that there has been an occasional dull ache after exertion at the right costal margin. The substernal radiating pain disappeared after the operation and has not returned.

Examination by Dr. Murphy on September 2, 1915.—There is some relaxation of the abdominal wall, and an incisional hernia is present, facilitating palpation of the abdominal contents. There is considerable thickening of the margin of the liver. In the gall-bladder fossa the tissues are indurated and as hard as wood, and therefore a little suggestive of malignancy. This degree of hardness may be due to the damming-back of bile within the liver from total obstruction of the common bile-duct at some point in its course.

COMMENTS

Dr. Murphy (September 3, 1915): From the patient's history we learn that the jaundice had preceded the operation of cholecystectomy, and that it had cleared up after operation, but returned as soon as the drainage-tube was removed.

The questions that come up in our mind are, whether this postoperative jaundice is due to blockage of the common duct by cicatricial constriction; to the escape of a calculus into the common duct at the time of the previous operation; or whether it was due to the presence of a calculus impacted in the common duct at the time of operation, which calculus was not detected at operation. There was no colic after the operation, but the jaundice returned just as soon as the drainage-tube was removed, and after the sinus had closed. The patient does not know whether or not calculi were removed at the time of the operation, and there is no record that calculi were removed from the common duct. The patient states that he himself opened and examined the gall-bladder specimen, and that it contained material resembling soft coal. Such a material is composed of cholesterin stained with bile. Remember, 98 per cent. by weight of biliary calculi consists of cholesterin, which is formed by epithelial-cell débris from the mucosa lining the gall-bladder. This inspissated bile is putty-like in consistence, and forms calculi by the process of crystallization. The precipitation of a few cholesterin crystals means that all the inspissated bile will crystallize, just as when syrup, after reaching a certain density, crystallizes completely after the addition of a few crystals.

OPERATION

[Incision through scar of previous operation.] You find the same condition here that you find in every incisional or postoperative ventral hernia—adhesions throughout the sac of the hernia. I must first try to dissever these adhesions in order to be able to recognize the anatomic landmarks. . . . Now for the first time I have gained access to the free peritoneal cavity, below the mass of adhesions.

There is the duodenum, you see. . . . My exploring finger encounters a calculus impacted in the ampulla of Vater. Whether I shall be able to remove the calculus by mobilizing the duodenum and dislodging the calculus by milking the duct from behind (subduodenal route), or whether I must, in addition to mobilizing the duodenum, cut through its anterior wall (transduodenal route) to remove the calculus, is problematic. . . .

I find that I must approach the calculus by the transduodenal route. [Incises longitudinally anterior wall of duodenum opposite site of calculus impacted in ampulla, nicks upper border of ampulla, and extracts calculus (Fig. 301).] . . . I am now closing the incision in the posterior wall of the duodenum with one continuous linen suture, and that in the anterior wall with three rows of sutures, the first being a Czerny suture for the mucosa, and the other two being of the Lembert type. [Inserts rubber tube for drainage.] In closing the wound I shall endeavor to correct that hernial protrusion of the anterior abdominal wall by a plastic operation. The width of the gap is too great to

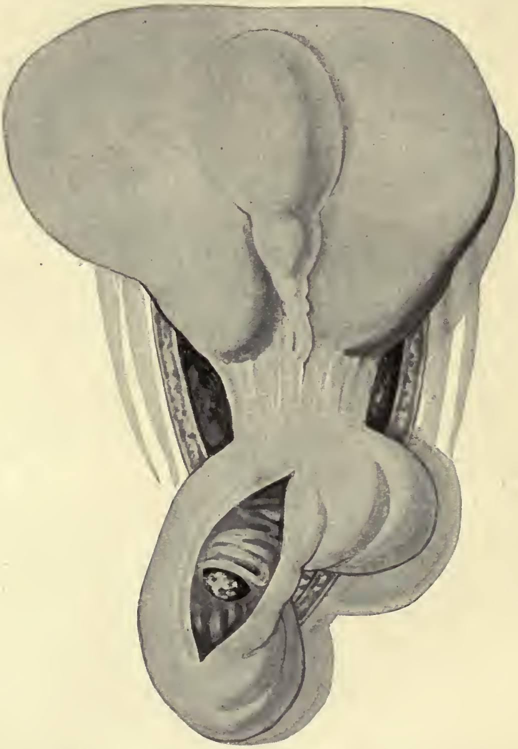

Fig. 301.—Duodenocholedochotomy (McBurney's operation). Stone in the ampulla. The edge of the ampulla is incised and the stone extracted (Moynihan's "Abdominal Operations").

permit an imbrication operation. [Isolates layers of abdominal wall from cicatricial mass.] I am putting in a double row of (figure-of-8) sutures—one layer including the peritoneum, the transversalis fascia, and the posterior sheath of the rectus muscle, and the other layer including the anterior sheath of the rectus.

RECAPITULATION

Let the record show that, after dissevering adhesions, we mobilized the duodenum by cutting the posterior parietal peritoneum 1½ inches to its outer side, and then swung the duodenum over toward the midline; that next we applied light intestinal clamps to the duodenum above and below the site of the impacted calculus, incised the anterior wall of the duodenum opposite the ampulla of Vater, and then incised the ampulla and extracted the calculus; that much of this work was extraperitoneal in the sense that it was performed outside of the free peritoneal cavity, and outside of the line of adhesions; and finally that, after reconstructing the abdominal wall, a rubber tube was inserted for drainage.

Let the record show also that Sister Laurian reported the sponges counted and correct; and that in closing the abdomen the operator inspected the field to see that no sponges or instruments were left remaining behind.

POSTOPERATIVE COMMENTS

The patient will have no food at all by mouth for four days: the stomach must be kept absolutely empty. We shall start proctoclysis immediately, giving 20 grains of the lactophosphate of calcium every four hours to accelerate coagulation of the blood. The fluids of the clyster serve to dilute the toxins in the blood. We shall leave these sutures in for from two to three weeks: in addition to the support derived from the sutures, the patient will have mechanical support of the abdomen, first by a bandage, and later by a binder.

We may now correlate the history with the operative findings. Although the patient was jaundiced at the time of the previous operation, yet there was no record of an obstruction having been removed from the common duct. While cholecystectomy was performed, yet removal of the gall-bladder did not dislodge the calculus obstructing the common duct below. As soon as external drainage of bile stopped from closure of the sinus, the jaundice returned: this return of the jaundice is explained by the presence of the calculus blocking the common duct—the bile could escape from the sinus so long as the sinus was open, but when the sinus closed, the bile could pass only into the vascular system and cause jaundice.

I should have preferred removing the calculus by approaching the duct from behind,—the subduodenal route,—but when the calculus has passed down the whole length of the common duct and becomes impacted in the ampulla of Vater, the plan of transduodenal removal, suggested by McBurney and adopted later by Moynihan, seems to be the best.

Now let me review the technic of the transduodenal route of removing a calculus impacted in the ampulla of Vater, as illustrated by this case. First, we separated the adhesions, beginning at the pylorus and working from the pylorus down to the duodenum, until we were able to expose the duodenum. When the duodenum had been exposed, we found that the calculus was situated directly behind the descending portion of the duodenum and a little to its median side. As you know, the second and third portions of the common bile-duct pass first behind the duodenum and then to the median side; and after the bile-duct is joined by the pancreatic duct, it passes downward and a little outward, so that the ampulla of Vater lies behind the descending duodenum and a little to its inner side. In order to remove a calculus from the ampulla of Vater by either route—the transduodenal or the subduodenal—it is essential that a certain method be followed: this method consists in dividing and freeing the posterior parietal peritoneum 1½ inches to the outer side of the descending duodenum, and then lifting the mobilized duodenum and throwing it over to the left, exposing the ampullary portion, after the plan suggested by Haasler at the German Surgical Congress in 1898. The duodenum was then isolated by gauze packs, and the light intestinal clamps were placed on the duodenum above and below the area to be incised, so as to prevent the admission of intestinal contents into the operative field. Then the anterior wall of the duodenum was incised and the calculus exposed, protruding through the posterior wall at the ampulla of Vater. The edge of the ampullary orifice was then nicked and the calculus extracted. Grasping the posterior wall of the duodenum with intestinal forceps, we sutured the incision with silk or chromicized catgut, and then sutured the anterior wall after the Czerny-Lembert plan, using an additional row of Lembert sutures.

[*N. B.*—The jaundice began to subside immediately after the operation. The wound healed by primary union, and the patient made an uninterrupted recovery.]

[In conjunction with the above case the following cases of biliary calculi, published in previous numbers of the CLINICS, illustrate fairly completely the *living* pathology and sequelæ of this malady: "Cholelithiasis," June, 1912, pp. 417–428; "Removal of Enlarged and Dilated Stump of Gall-bladder Following a Previous Operation, with Secondary Perforation of its Wall by Three Calculi," February, 1914, pp. 173–178; "Cholelithiasis; Pericholecystitis; Stones in Cystic Duct; Cholecystectomy," April, 1914, pp. 237–245; "Cholelithiasis; Calculi in Common Duct, with Intense Jaundice; Talk on Cholelithiasis and Cholecystitis," December, 1913, pp. 1043–1059; "Obturation Ileus; Obstruction Due to Large Gall-stone in Ileum," June, 1913, pp. 353–370; "A Diagnostic Talk on Intestinal Obstruction Due to a Large Gall-stone," June, 1915, pp. 447–456; "Cholelithiasis—Acute Hemorrhagic Pancreatitis—Incision and Drainage; Pancreatic Cyst—Deferred Marsupialization of Cyst," August, 1915, pp. 751–759.]

ADENOCARCINOMA OF NECK OF UTERUS—VAGINAL HYSTERECTOMY BY JACOBSON'S METHOD

Summary: Clinical history of patient; vaginal examination; comments—differential diagnosis from senile vaginitis, submucous polyp, and carcinoma of fundus; without the presence of saprophytes there is no intrinsic odor to a cancerous discharge; operation; postoperative comments—vaginal hysterectomy safer than supravaginal and not attended by the complications of the latter.

CLINICAL HISTORY OF PATIENT

THE patient, a married woman aged sixty years, was admitted to hospital June 3, 1915. The family and past histories are negative. The menopause occurred fifteen years previously, when the patient was forty-five years old.

Two years ago, in 1913, after a period of housecleaning, the patient noticed for the first time a blood-tinged vaginal discharge which lasted one day. The discharge was not associated with pain, nor with a bearing-down sensation. Six weeks later she again noticed a blood-tinged vaginal discharge, and thereafter this phenomenon appeared every six weeks until October, 1914, when it became almost continual, and at intervals within the last few weeks previous to admission pain has been associated with the discharge. There has never been a severe hemorrhage, and at no time did the amount of the flowing equal or surpass that of a normal mensis. The patient's weight has shown no significant decrease.

Vaginal examination reveals that the uterus is displaced posteriorly, and that the fundus is fixed by adhesions in Douglas's pouch. The cervix is small, soft, and the seat of a slight erosion, which has not, apparently, penetrated the underlying tissues. Sponging or even digital examination makes the erosion bleed. There is no relaxation of the perineum from laceration.

COMMENTS

Dr. Murphy (June 4, 1915): If the amount of the flow did not equal or surpass that of a normal mensis, there are three possibilities in the differential diagnosis. The first possibility is senile vaginitis: in this disease the vagina is small and bleeds upon the slightest contact, and this tendency to hemorrhage often lasts for years. The second possibility is a submucous polypus of the uterus, which, when present, maintains a serosanguineous discharge for years; and as the uterus contracts it frequently expels the polypus from the uterine cavity into the vagina—an event that is accompanied by uterine colic and slight bloody discharge. The third possibility is carcinoma of the fundus of the uterus. The history states that upon one occasion there was a very offensive leukorrhea: I have never seen an offensive leukorrhea due to carcinoma that, once established, did not continue. The fact that the discharge subsided, then, leads us to the conclusion that there is some lesion of the uterus other than a carcinoma that is causing the bleeding.

Carcinoma of the fundus of the uterus usually gives rise to a hemorrhage which is characteristic in that, a year or more after the menopause, it suddenly appears as a profuse flow, surpassing in amount that of a normal mensis. A similar profuse hemorrhage may or may not recur. If it does recur, it reappears within a short time as a serous or serosanguineous discharge that may have an offensive odor: the rule is that there is no offensive odor until saprophytes have been implanted in the vagina by the manipulations consequent upon examination, or by the introduction of a foreign body into the vagina. Without the presence of saprophytes there is no intrinsic odor to cancerous discharge.

In this case examination reveals an erosion of the cervix; but this erosion does not resemble one due to carcinoma. The presence of carcinoma of the fundus cannot be detected by vaginal examination: the fundus, however, is softened and larger than it should be at this patient's age.

OPERATION

[Circular incision through vagina close to cervix—bladder and parametrial tissues peeled back from uterus—fundus drawn down into vagina.] You will observe that now I have the whole uterus down and clearly in view, so that I can clamp the broad ligaments on each side with these long, 8-inch clamps. . . . This clamp-method, which we used originally in performing vaginal hysterectomy, is seldom employed by us at present. We surround the clamps with two strands of nosophen gauze secured by a safety-pin, and shall let the clamps remain in place for seventy-two hours. The clamp-method gives rise to a little more postoperative pain than the suture-method.

POSTOPERATIVE COMMENTS

From a mortality standpoint this operation of vaginal hysterectomy, so strongly advocated by Jacobson, is the safest—1 per cent., and when performed for cancer, from 2 to 2½ per cent. —while the Wertheim operation has an operative mortality of 25 per cent., and an operative morbidity of 11 per cent. due to such complications as vesicovaginal or rectovaginal fistula or necrosis of the bladder or ureter.

I just had in my office to-day a patient upon whom vaginal hysterectomy by the clamp-method had been performed three years and one month ago. There is not the slightest evidence of recurrence. The vaginal operation for cancer of the uterus I believe will be resorted to as the safest and, on the whole, the most life-conserving procedure: First, because it has an operative mortality of only 2½ per cent.; secondly, it has only a very small percentage of operative morbidity—the exact percentage cannot be obtained; thirdly, the operative incapacity is short, and the patient's resistance is not materially lowered by the operative onslaught; finally—and most important—our analysis of over 3000 postmortem reports of examinations for metastases shows that 30 per cent. of the patients who die from carcinoma of the uterus have no metastases anywhere in the body, not even in the lymph-nodes. Therefore the removal of the latter is not necessary in 30 per cent. of the cases, even when the disease had progressed to the fatal stage. The procedure, therefore, that has the least operative mortality and operative morbidity is the one that should be adopted. In cancer of the uterus we believe that such a procedure is vaginal hysterectomy by the clamp or suture method.

[This patient made an uneventful recovery without operative morbidity.]

[*N. B.*—Microscopic examination by the pathologist, Dr. Herzog, revealed adenocarcinoma.]

[A table of statistics of uterine carcinoma was published in the Clinics for October, 1915, p. 917. The following note is appended, supplementary to that table:

In a paper entitled "Zur erweiterten vaginalen Karzinomoperation" (*Zentrbl. f. Gynäkologie*, October 16, 1915, No. 42—Abstr. in *Jour. Amer. Med. Ass.*, November 20, 1915, 1860), Thaler brings the record down to date, the total number of cases of uterine cancer at Schauta's clinic, between 1901 and 1914 inclusive, being 1305. In 654 an operation was performed by Schauta's "extended" technic, and 133 of the women have been cured with no signs of recurrence for five years at least. This is a total proportion of 38 per cent. permanent cures. Computed according to Winter's scheme, this is an absolute cure of 17.4 per cent. of all the cases seen, and, restricting the figures to 1905–1909, we have 20 per cent. absolute cures.]

UNDESCENDED TESTICLE—ORCHIDOPEXY; INGUINAL HERNIA — HERNIORRHAPHY (TWO CASES)

Summary: Clinical history of patients; comments—discussion of subject of undescended testicle; description of a case of pseudohermaphroditism; case in which testicle remained in abdomen and underwent sarcomatous degeneration; metastasis of sarcoma to organ of opposite side after removal of one sarcomatous kidney, testicle, or ovary; the Cohnheim *vs.* other theories for the development of malignancy; the significance of the scrotum and dartos; operations; recapitulation; postoperative comments—treatment of undescended testicle by administration of desiccated thyroids.

CLINICAL HISTORY OF PATIENT

The patient, aged thirty-nine years, married, was admitted to hospital December 30, 1914. The family and past histories are negative.

The present absence of the right testicle from the scrotum was noticed at birth. There was also a reducible inguinal hernia on the same side. Until he was fifteen years of age the patient wore a truss and then discarded it. During the past five years, however, he wore the truss continually. At present the hernia bulges, transmits an impulse on coughing, and is with difficulty kept reduced.

COMMENTS

Dr. Murphy (December 31, 1914): Of the cases of undescended testicle, there are first those which become arrested at some point in the normal path of their descent. The *anlage* of the testis lies opposite the lower pole of the kidney; the organ may remain at this site and never descend at all. It may pass down behind the peritoneum, and, from arrested descent, remain in the abdomen, the inguinal canal, or just outside of the external abdominal ring: and, if misguided by the gubernaculum, it may lodge in the perineum, over the symphysis pubis, or near the anterior superior spine of the ilium, and may even pass down and lodge superficially in the thigh.

In the ectopic varieties the testicle may remain in the pelvis, and, as in one of our cases, it may become attached to a Fallopian tube and be drawn by the tube in its descent down into the pelvis. In the patient we refer to—a pseudo-hermaphrodite—both the vagina and the penis were fairly well represented. We could pass a probe through the sinus that represented the small vagina up into the well-developed uterus. Both Fallopian tubes were developed; and at the fimbriated end of each tube there was a testicle instead of an ovary. In place of descending extraperitoneally toward the inguinal canal, the testicle went directly from its position at the lower pole of the Wolffian body over to the Fallopian tube, to which it became attached. One of these testicles had undergone sarcomatous degeneration of the round-celled type. We believed—rather, we were taught to believe—that an undescended testicle underwent sarcomatous degeneration from the trauma to which it was subjected when slipping in and out of the inguinal canal, but in this case the sarcomatous testicle was free in the peritoneal cavity, and not subjected to traumatic irritation. We removed the sarcomatous testicle, but permitted the other one to stay *in situ:* no attempt was made to implant the remaining testicle for fear of the trauma to which it would have been subjected in its transplanted position, possibly leading eventually to sarcomatous degeneration; and then we did not want to unsex the lad entirely. At the close of the operation we placed two artificial testicles in the scrotum. Along the vas deferens there was a portion of the epididymis which hung out very much like an ovary: microscopic examination proved its testicular structure. Even at operation it was apparent that the organ was a testicle and not an ovary.

The chances of that undisturbed testicle becoming sarcomatous are great, because when sarcoma develops in one testicle and that testicle is removed, a similar malignant change takes place in the remaining testicle. The same cycle obtains for the kidney and the ovary. The order of frequency of the metastasis of sarcoma to the opposite side in these paired organs after removal of the one primarily involved is first the kidney; secondly, the testicle; thirdly, the ovary. In his paper on the "Kidney," which appeared in 1896, Morris stated that he had never seen permanent cure follow nephrectomy for sarcoma. Most of the permanent cures reported in the literature have followed nephrectomy for hypernephroma. These factors could lead one to believe that the Cohnheim theory of the development of neoplasms is correct, but we are drifting away from the Cohnheim theory and inclining toward the belief that the malignancy of cells of sarcomata and carcinomata is due to the influence of some biochemical agent. The question arises whether or not this biochemical agent is implanted in its position in the embryonal state and remains quiescent until later years, just as when the spirochete, implanted in the embryonal state, begins to exert its characteristic destructive influence many years afterward. We know that numerous anomalous conditions which are peculiar to the embryo, but persist after birth, do not manifest themselves at all until late in adult life. Such a condition is cervical rib, which does not usually manifest itself until after the thirtieth year, on an average (*vide supra*, p. 1069).

For many years I have had under observation a patient one of whose testicles comes out directly through the internal abdominal ring and passes on to the anterior abdominal wall, between the muscular layers of which it lies, in a serous pocket extending up to the anterior superior iliac spine. Associated with the undescended testicle in this case there is a small hernia—so small that it has caused the patient little or no trouble. He refuses to have the congenital deformity corrected. In such a type the testicle remains high up, while the dartos is developed below; and in another type the testicle remains high up, but no dartos is developed below.

What is the anatomic structure of the scrotum? It is the continuation of all the layers of the abdominal wall with the muscle-fibers almost entirely absent (Fig. 304, s). The dartos of the scrotum represents the thinned-out deep layer of the superficial fascia of the abdominal wall, carried down from the external abdominal ring. The cases of undescended testicle in which the dartos is not developed present a much more difficult proposition to deal with than those in which the dartos is developed and already down in the scrotum. The tunica vaginalis, of course, represents merely the peritoneal pouch in the scrotum, which bears a double capsular relation to the testicle.

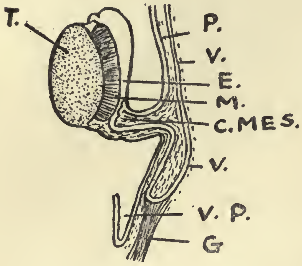

Fig. 302.—Showing elongated common mesentery of testis. T, Testis; P, peritoneum; V, vas; E, epididymis; M, mesorchium; C, common mesentery; V. P., vaginal process; G, gubernaculum (A. C. Wood, after Keith).

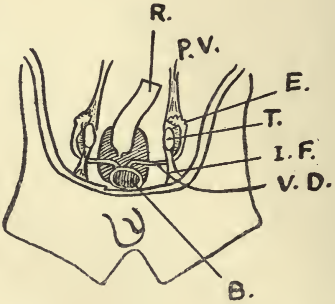

Fig. 303.—Fetus of two and one-half months. R, Rectum; P. V., plica vascularis; E, epididymis; T, testis; I. F., inguinal fold; V. D., vas deferens (A. C. Wood, after Keith).

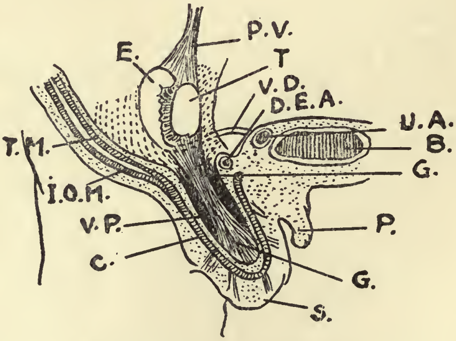

Fig. 304.—Fetus of sixth month. P. V., Plica vascularis; E, epididymis; T, testis; V. D., vas deferens; D. E. A., deep epigastric artery; U. A., umbilical artery; B, bladder; P, penis; G, gubernaculum; S, scrotum; T. M., transversalis muscle; I. O. M., internal oblique muscle; V. P., vaginal process; C, cremaster (A. C. Wood, after Keith).

OPERATION

[Incision made to expose right inguinal canal.] There is the properitoneal fat presenting; here is the hernial sac. [Opens sac.] There is the gubernaculum testis, trying to bring the testis down. . . . Here is the testis, behind the peritoneum. Note that the vas deferens is not attached to it below; that is by no means an uncommon condition to encounter; on following the vas upward, however, we find that it is attached to the testis above, at its upper pole. Here are the vasa efferentia—the important efferent ducts of the testis. The epididymis here is plicated with the vas deferens below the level of the external abdominal ring. Now we must try to free the testicle and get it down into the scrotum.

There is the peritoneal sac, which corresponds to the vaginal process (Fig. 302, V. P.), and which is spread out as a large pocket in the anterior abdominal wall, just beyond the pillars of the external ring. Now we shall shell this peritoneal sac out from its interparietal position, using the same method as that employed in shelling the tunica vaginalis out of the scrotum. I am going to free this peritoneal pocket so as to be able to deal with the spermatic cord. . . . I am freeing and isolating the vas deferens and the vessels of the cord from this portion of the peritoneum. . . . I have succeeded in separating this peritoneum from all the structures that, with the vas deferens and the associated vessels, compose the spermatic cord: all the vessels are now behind the peritoneal pouch. There is the deep epigastric artery pulsating in its proper relationship, internal to the cord (Fig. 304, D. E. A.). There is the vas deferens as it comes up from the epididymis, just behind the peritoneum (Fig. 304, V. D.): I am freeing it so that the testis may be brought down without too much tension upon its vessels. . . . At last we have freed the testicle and are ready to place it in the scrotum.

The gubernaculum (Figs. 302, 304, G) is not attached to the testis: it is attached to the epididymis, which was dragged down by the gubernaculum while the testis remained up above.

Now we shall prepare the new scrotum for the reception of the testicle. There is the dartos tissue, over the symphysis pubis. [Dissects up flap containing dartos, stretches it, and pushes it downward so as to form a new lining for the scrotum; testicle placed in scrotum.] I am fixing the most dependent portion of this testicle in the scrotum so that the organ cannot retract into its old position. We anchor it to the connective tissue of the scrotum by means of a few catgut stitches, and, in addition, by passing two horsehair sutures from the skin surface into the scrotum and through the connective tissue at the lower pole of the testicle (Lanz's method).

Now we are ready to reconstruct the inguinal canal, and we shall employ the imbrication operation of Andrews, which corresponds to the overlapping flap method of Mayo for umbilical hernia. [For an illustrated description of this operation, edited by Dr. Andrews, see the CLINICS for October, 1913, pp. 756–768.]

RECAPITULATION

Let the record show that, after making the incision, we freed the testicle and opened the peritoneum; that we separated the vas and vessels from the peritoneum well down until the testicle was perfectly loose and mobile; that then the scrotum was prepared and a pocket made in it for the reception of the testicle; that the scrotum was dilated with an 8-inch angiotribe; that the testicle, now perfectly free, was displaced into the newly formed scrotum, to the bottom of which it was secured; that the cord was brought down beneath the aponeurosis of the external oblique, which was overlapped by the imbrication plan; and that when the operation was completed there was no tension on the vas or vessels. No sponges were used in the peritoneal cavity, and therefore no record of them is made.

POSTOPERATIVE COMMENTS

I shall probably have an opportunity this week of showing a patient in whom we made a new dartos. The operation was performed many years ago. When we saw him originally there was nothing at the normal site of the scrotum but skin—no sign of any dartos. Operating through a curved incision, we formed from the suprapubic displaced dartos a pocket that corresponded to a new dartos in the scrotum, for the protection of the testicle.

CASE II

Clinical History of Patient.—The patient, aged thirty-six years, single, was admitted to hospital January 30, 1915. The family history is negative. Eleven years previously, in 1904, he was operated upon for right-sided inguinal hernia.

Absence of both testicles from the scrotum was noticed at birth. At the age of fourteen years the left testicle descended spontaneously into the scrotum, and at present it is freely movable therein. The left side is free from hernia.

The right testicle has never descended into the scrotum. When the hernia on the right side descends, if the testicle goes ahead of the hernia, it causes the patient great pain; but if the intestines go ahead of the testicle, the pain is not so severe: the patient can tell by the degree of pain which organ is in advance. The hernia bulks most when the patient is standing up, but it is readily reduced. The patient wore a truss for one year before the first operation. The hernia has never descended completely into the scrotum.

This right-sided undescended testicle has already been operated upon (1904). Half of the right testicle was resected at the time of the operation, but the organ itself was not transplanted into the scrotum. The hernia recurred one month after the operation, but is still easily reduced. Ever since the recurrence of the hernia the patient has been wearing a truss. When the hernia is down, there is no longer any pain, and the bulging is still more marked in the standing position.

Comments.—Dr. Murphy (February 1, 1915): In this case I do not know what conditions I am going to encounter upon exposure, because I do not know what disposition of the testicle was made at the time of the first operation. Testes that do not come out of the abdomen do not at first have the epididymis attached to them. Normally, the epididymis precedes the testis in its descent (Figs. 302, 303, 304, E). The vaginal process descends attached to the gubernaculum testis (Figs.

302, 304, V.P., G), and finally the testis comes down with the peritoneum, and after that the epididymis becomes fixed to the testis. In this case we do not know at what point in its descent the testicle has been arrested. The hernia has recurred since the first operation.

Operation.—Here we have to deal with the cicatricial tissue that developed in consequence of the previous operation. [Incision.] Now we are beginning to see the aponeurotic fibers of the external oblique muscle, as we cut down through the cicatricial tissue. . . . We must endeavor to find some additional landmarks in this scar tissue. . . . This tissue looks like some fibers of the internal oblique muscle, but its identity is obscured by the cicatricial tissue. . . . There is the testicle: it is fairly large in size. I am coming along the hernial sac and freeing it. . . . That looks like dartos tissue. . . . Now we must get the peritoneum free. . . . Upon opening the peritoneum of the sac we find that it contains omentum, which must be reduced into the abdominal cavity.

Let the record show that there were no adhesions in the sac except at one little point.

The next step is to get the peritoneal sac free from the vas deferens and vessels: it has never been freed from these structures, and the presence of the cicatricial tissue, which resulted from the previous operation, makes the freeing process more difficult. . . . There is the vas deferens—away to the inner side: it is irregular in its disposition, and does not pull out in the usual manner. . . . I am now freeing the peritoneal sac backward as far as the internal ring. . . . I am pulling the vas still farther down, so that I can ligate the sac and not include the vas. . . . I am looking again to make assurance doubly sure that I have not included the vas in this ligature. . . . I am now tying the stump of the sac very firmly—tightly enough to occlude it. After ligating the sac, using the same suture I anchor the sac to the margin of the internal ring so that it cannot possibly get away. . . . Now I want to release the fibrous tissue around the vas and the blood-vessels, so the latter structures may be brought down without undue tension. . . .

The testicle has at last been sufficiently released from the fibrous tissue so that it can be brought down into the scrotum without any tension upon the vessels.

If I do not reconstruct the inguinal canal, I shall not succeed in curing this hernia. . . . There is the deep epigastric artery pulsating in the field. . . . From what I find here I take it that this was originally an interparietal hernia, because the tunica vaginalis has passed into the anterior wall of the abdomen. The surgeon who operated upon this patient previously lost his bearings, but was wise enough to know when to quit—that was good judgment on his part. The hernia, of course, recurred, because the canal was not properly closed. So rare is this condition of undescended testicle, that a surgeon with a limited experience may be excused for not recognizing the situation. [Testicle drawn down and placed in a pouch made for it in the scrotum, and then anchored. Inguinal canal reconstructed by Andrews' method, as described with the previous case.]

Recapitulation.—Let the record show that the testicle was well developed, but the epididymis was not attached to it in the usual manner, but was represented by an elongated continuation of the vas deferens; that the vas and blood-vessels were found and isolated from the peritoneum; that the testicle was first exposed and next the hernial sac, which was ligated and disposed of in the usual way; that the testicle was separated from the peritoneum, passed into the scrotum, and secured to the most dependent portion of the scrotum by the regular technic.

Postoperative Comments.—Within three weeks this bulging of the inguinal region will have practically all disappeared, until the normal size has been reached. With the subsidence of the swelling the testicle will remain securely in its regular place in the scrotum. In some cases of imperfectly descended testicle the processus vaginalis is found in the scrotum, but the upper end of the processus may not become shut off from the general peritoneal cavity, and there is always a potential or an actual inguinal hernia. Fluid may collect in the peritoneal pouch and form a congenital hydrocele.

The diagnosis of undescended testicle is usually easy, and is made by finding the scrotum empty, and palpating the organ in its abnormal position, provided that it has not remained within the abdomen.

Operation is indicated in all cases, except in those in which the testicle is situated within the abdomen. The organ may be fixed in the scrotum, removed from the body, or returned to the abdomen.

Armstrong, of Wellington College, reports remarkable results from the administration of thyroid extract in cases of undescended testicle (*Med.-Press. and Circ.*, August 4, 1915). In this preliminary note he says: "During the last four years, out of about 600 boys of the age of thirteen to fourteen years who have presented themselves for examination, on entry 4 have been found with complete absence of both testicles from the scrotum. In each case the penis was very small, and there was no appearance of hair upon the pubis. Two of them had Levi E. de Rothschild's sign of thyroid inadequacy—*i. e.*, rarefaction of the outer third of the eyebrows. One boy was of the Mongolian type.

"To each of them thyroid extract in half-grain doses was administered twice a day over a considerable period, with satisfactory results. The effect of the treatment was almost immediately apparent; in a few weeks the testicles could be felt in the inguinal canal, and in 3 of the 4 cases the complete descent of the testicles into the scrotum was established in about three months. In the fourth case, which is still under treatment, one testicle, the left, is completely and the other almost descended.

"In the first 2 patients, who are now seventeen years of age, the growth of pubic or other body hair is still very scanty."

[Previous articles upon the subject of undescended testicle have been published in the CLINICS as follows: December, 1913, pp. 1033–1042, "Undescended Testicle in Inguinal Canal" (embryologic, anatomic, and clinical phases); April, 1914, pp. 217–236, "Three Cases of Ectopia Testis" (pathologic conditions, embryologic diagrams, and illustrations showing steps of operation).]

CONGENITAL LUXATION OF BOTH HIPS—METASTATIC ERYSIPELATOUS BILATERAL EPIPHYSITIS OF HEAD OF FEMUR—BLOODLESS REDUCTION OF BOTH LUXATIONS

Summary: Clinical history of patient; comments—changes wrought in hip-joints by former erysipelatous infection; pathologic separation of upper epiphysis of femur; pathologic anatomy of congenital hip luxations; operation—tenotomy of adductor tendons; reduction of left side; mechanism of reduction; analysis of Lorenz method; reduction of right side; test of reduction; position in which limb is fixed after reduction; dressing.

CLINICAL HISTORY OF PATIENT

THE patient, a female aged three and one-half years, was brought to hospital October 1, 1914. The family history is negative. The mother was in labor twelve hours. No instruments were used in the delivery. The mother noticed no deformity of the child at birth.

The present trouble was first noticed when the baby was five weeks old, and began with an attack of erysipelas, which, commencing in the groin, spread around the pelvis, the lower one-third of body, and the lower limbs. During the fourth week she developed abscesses, one on each hip and on the chest, head, and ankles. All these abscesses were lanced, and much pus was evacuated. In the fifth week she had thrombophlebitis of both legs. She was in bed seven weeks altogether.

At the age of two years, when the child began to walk, the mother noticed a peculiarity in the gait. At present the gait is waddling and rolling from side to side. The buttocks seem prominent, and the hips and perineum appear broader (Fig. 305). In walking, marked lordosis appears. The mother says that the child does not seem to suffer pain.

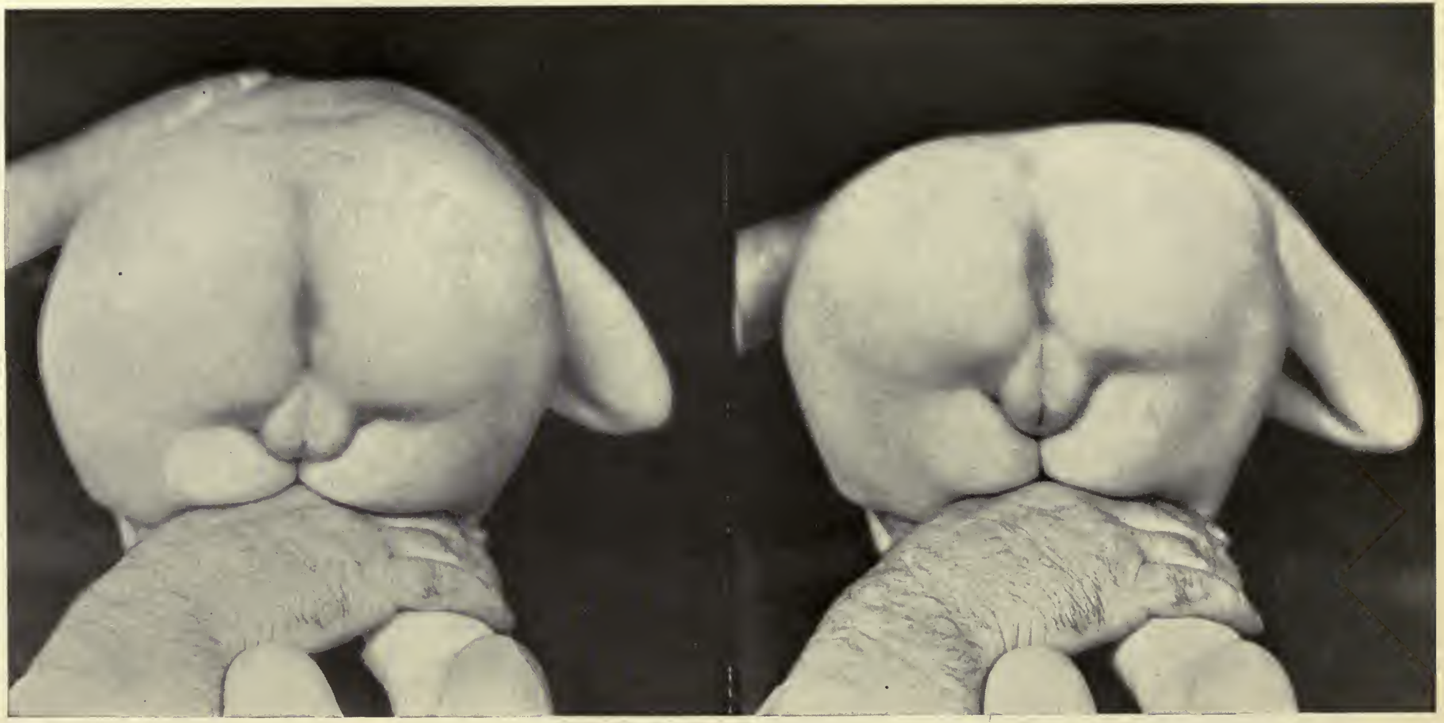

Fig. 305.—Preoperative photograph of double congenital luxation of hips. Note prominence of buttocks and broadening of hips and of perineum.

COMMENTS

Dr. Murphy (October 3, 1914): This is the case that we showed last Wednesday. We have had more skiagrams made, and Dr. Porter will tell you his conclusions.

Dr. Porter: When Dr. Murphy and I saw the case we had no history of anything except of the present condition, being told that the child was as late in walking as the age of eighteen months. The patient was brought here for reduction of the deformities, and Dr. Murphy requested me to attempt to reduce the luxations. We had some skiagrams of the hip and of the pelvis taken. The morning that the child was brought into the operating-room the skiagram was placed in the shadow-box. Upon looking at the picture I said to Dr. Murphy that we were dealing with something more than congenital luxation of the hips. That picture did not show an ordinary congenital luxation. There, from another case, is the picture of an ordinary congenital luxation of the hip on one side only. In that case the luxation was reduced four or five weeks ago. In it you see the sharp outline of the acetabulum on this side, and capping the upper end of the femur the center of ossification, the epiphysis. Following out from the epiphysis you trace a clean-cut, sharp outline. On the other hand, when you look at the picture of this patient before us (Fig. 306), you see the acetabula appear rough and eroded, as though there had been some inflammation in the hip-joints. The upper ends of the femora cast no shadow: you can see no centers of ossification. Altogether, the joints look as though they had been involved in some infection, inflammation, or other condition that changed the normal appearance of the congenital luxations. Puzzled, we then learned the rest of the history.

Now, were this patient presented to you without any history whatsoever she would pass muster as a case of ordinary double congenital luxation of the hips. We shall demonstrate to you the wide pelvis, the prominence of the femora, and the breadth of the perineum (Fig. 305). The fact that the child did not walk until she was almost two years of age is another point in the history that goes to make up a beautiful clean-cut picture of a congenital luxation of the hip, but the appearance of that *x*-ray picture is one of the things that changed the diagnosis. I have been doing this work for twenty years, but have yet to see a congenitally luxated hip that presented such a picture. It is unique (Fig. 306).

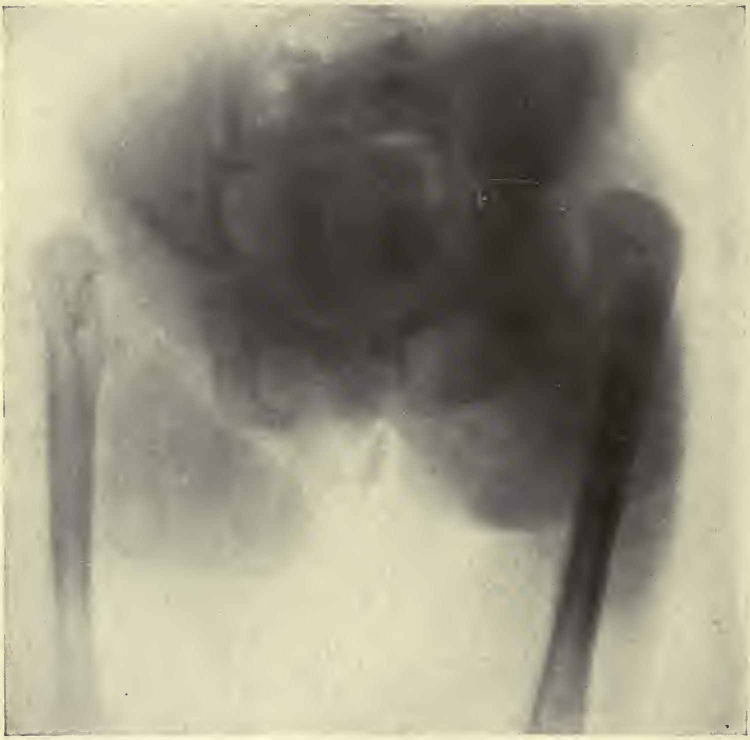

Fig. 306.—Preoperative skiagram showing congenital luxation of both hips. Note roughening and erosion of acetabula. On right side the epiphysis has been displaced; on the left side the epiphysis has been destroyed (anteroposterior view).

I am convinced that the child was born with congenital luxation of both hips, but at the time that the erysipelatous infection occurred there was some inflammation in this hip-joint by extension or metastasis which affected the epiphysis at the upper end of each femur, for the upper end of each femur is diseased. Here is another picture that was taken since the child appeared in the clinic [demonstrates]. On the right side you can see that the center of ossification is apparently displaced to between the upper end of the femur and the lower part of the acetabulum, instead of being directly over the neck of the femur. It looks as though the epiphysis had slipped off, as is the case in so many infections of the hip-joint. Take, for instance, typhoid infection of the hip-joint: in typhoid infections the epiphysis is the site of the infection, and the epiphysis becomes either completely destroyed or permanently distorted. On the left side you can see nothing of the center of ossification of the upper end of the femur. The area corresponding to the neck has been disturbed.

Upon careful physical examination of the patient you notice at once that there is not the freedom of motion in the upper end of the femur that there is in the ordinary single congenital luxation of the hip. In congenital luxations of the hip all movements at the hip-joint are usually preternatural and free, except the movement of abduction, which is limited on account of the shortened adductor muscles on the inner half of the capsule. Upon examining this patient the motion is not so free. You can move the upper end of this femur as freely as you can move a normal hip—indeed, you can move it more freely than you would have expected in a case that had become infected. In palpating the head of the femur behind the acetabulum you find the enlarged upper end of the femur, but not the round, smooth head that you can palpate in the usual case of congenital luxation. And so, after seeing the new *x*-ray pictures and after talking to the parents, I said that I would attempt to reduce the luxations and at the same time divide the adductor longus tendon, which is still the obstacle to lifting the upper end of the femur into place over the border of the acetabulum, and that I would try to stretch the tissues in relation with the inner half of the capsule to hold the head in place. So, in the future, when the gypsum case is changed, we can complete the reduction with additional manipulation if we are unable to effect reduction at one sitting this morning.

Dr. Murphy: I did not figure that the erysipelatous infection had extended over into the acetabulum.

Dr. Porter: The right limb, when measured, is a little shorter than the left. This difference in length is due to the fact that the left femur has retained a little more of the epiphysis, and thereby has been able to outdistance its fellow in growth. Let me show you the scars left after the erysipelatous infection. Here is one in front; here is another over this right hip, and there is one over the left hip. There have been foci of suppuration everywhere which were due to erysipelas. A local erysipelas does not extend as deeply as the capsule. Involvement of the capsule and of the joint indicates metastatic hematogenous infection from a distant focus, and not a direct lymphatic extension from a local focus, because there are no lymph-vessels that lead directly into the joint.

Dr. Murphy: Dr. Porter will try, by continued pressure, to get the head into the remains of that very shallow and badly deformed acetabulum, and to retain it there until it has united. If he cannot do that, he will have to perform an open operation.

Dr. Porter: This case interests me very much because an infection or some other cause has disturbed the normal condition of the luxated hip. In this picture [indicating], which is a classic luxation, you can see that on the left side the acetabulum is distinctly higher than the upper epiphysis of the femur. On the right side you see that the upper margin of the acetabulum is shallow and deformed, and that the epiphysis, which on this side lies opposite the acetabulum, is involved in an old process of disease. We routinely take *x*-ray pictures of these cases of congenital hip-luxation to show the condition of the acetabulum. In some cases the upper margin of the acetabulum is dimpled and promises to retain a replaced head: in this patient of ours the upper border is shallow. I had some doubt about being able to retain the head in place at the time the luxation on the right side was reduced. Sometimes we make two, three, or four attempts to reduce the luxation before we do the open operation. In the patient I referred to a few minutes ago it took about four minutes to reduce the luxation. The head went into place very easily, and you could feel through the groin the head of the femur in the acetabulum: from all appearances it ought to stay there. We maintain plaster-of-Paris immobilization for three years without disturbing the limbs.

Left Side.—We start with the upper end of the femur behind the acetabulum. The shaft of the femur will fall inward in adduction. The upper end of the femur slides upward upon the dorsum ilii and the adductor muscles shorten to accommodate themselves in the resulting diminished distance between their origins and their insertions. Abduction is the movement that is impossible, and yet abduction is the most essential motion to restore. I am simply going to tenotomize subcutaneously the adductor longus tendon on this side, thereby doing away with the chief obstacle not only to abduction, but also to lifting the head of the femur into the acetabulum. The other obstacle to reduction is the shortening of the inner half of the capsule.

First, I locate the adductor longus tendon beneath the skin with my index-finger, and follow this guide with a blunt tenotome introduced through a preliminary nick in the skin. Can you see the adductor longus tendon standing out like a violin-string right between my fingers? With a blunt tenotome the tendon is sawed right in two: I can feel it yield. Its division affords a free range of movement when we attempt to reduce the luxation. In an ordinary case of tenotomy we just seal the wound with collodion. Here we are going to manipulate the joint, and the danger of leaving that little wound open would be a real one, so that we shall close it immediately with a single stitch. Ordinarily we do not suture it.

Here is the anterior superior iliac spine. The upper end of the femur is opposite this spine, but usually you can make out with your fingers two distinct prominences. That is the great trochanter, at the upper end of the femur. On abducting the limb you put your thumb down over this hollow place opposite the acetabulum. As the head is reduced one feels it moving into the acetabulum and filling up the empty space that was due to the absence of the head. When the femur is strongly flexed upon the abdomen, the short pelvic muscles running from the pelvis to the trochanter act as tensors or fulcra about which the upper end of the femur rotates. The head moves first from above and behind the acetabulum down to below the acetabulum. With the head shifted that far it remains to abduct the femur and lift the head of the femur over the lower, outer quadrant of the shallow acetabulum into place. Now I shall stretch the capsule extensively.

Some one said to me: "Are you going to reduce the luxation by the Lorenz method?" I said, No. The plan in the method of Lorenz is to tear all the adductor muscles on the inner side of the femur. The last time I saw Lorenz reduce a hip-luxation by his method was in 1907, in Vienna. The patient was a little girl of about five years of age. All the adductor muscles were torn free from the pelvis. From Vienna I went to Munich, where I saw Lange employ the same method in a little child of about the same age. Lange, however, took the greatest pains not to tear tissue that did not have to be torn to effect reduction. When the upper end of the femur slid finally over the lower quadrant of the acetabular margin it was retained in place by very heavy traction, and the limb was rendered absolutely immobile.

When the head slips over the acetabular rim into the acetabular cavity you do not feel the distinct click that occurs in the reduction of a traumatic luxation, but there is a characteristic motion that is appreciable. In a child of this age we expect to feel the head slide into place within a few minutes. There! It now rests in the acetabulum. You see the hamstring muscles tighten up immediately. I could feel the head when it slid over the acetabular rim. This luxation on the left side was harder to manage because the joint was more extensively eroded upon that side, as the skiagram reveals.

Right Side.—Now we shall go to the right side. I can feel the upper end of the femur lying just below the acetabulum. I can feel the head slide past the acetabulum instead of sliding into it: therefore, we must stretch these adductor tendons so the head will slide into place. Though in the skiagram this looked like the better head, yet now it gives us the impression of being not so good as the head on the left side. In the usual congenital luxation we do not feel a distinct click during reduction, because, owing to the relaxation of the capsule, the head is brought down upon the round ligament simultaneously with the loose capsule: ordinarily, however, the round ligament amounts to nothing more than a little atrophic band of tissue.*

The age of the patient is not always indicative of the degree of difficulty to be anticipated in making the reduction. At the Home for Crippled Children we had recently a luxation of the hip-joint in a boy seven years of age, who was uniformly well developed and whose muscles were strong. I anticipated great difficulty in reducing the luxation of the hip-joint in his case, but the reduction was accomplished within a few minutes, and was accompanied by a distinct click that was audible to all standing around the operating-table.

A week later we had a patient who had double congenital luxation of the hip-joint. The patient was a little girl about four years of age. After manipulating for twenty minutes I was unable to reduce the luxation. I had as a guest Dr. Bradford, of Boston, one of the most capable orthopedists in the country. He, too, tried for twenty minutes to reduce it and also failed. In similar cases it is my practice to drag the head of the femur down as nearly into the acetabulum as is possible and then fix the child in plaster-of-Paris in this "frog" position, which is maintained for from ten days to two weeks. Then, at a later sitting, the reduction is more likely to be accomplished.

This head just slid a little and I could feel it click. In these luxations reduction is effected when least expected, and after you have been trying for a long time to get the head back into place. The proof of reduction is the palpation, through the groin, of the head of the femur in the acetabulum, with obliteration of the hollow place that was previously in front of the latter.

DRESSING AFTER REDUCTION

In applying the fixation-dressing, a stocking is first drawn over the limb, and over the stocking a plaster-of-Paris bandage is applied with turns about the abdomen. The limbs are flexed and abducted to the superlative degree—into the "frog" position of Whitman (Fig. 307, p. 1170). This position is maintained for three years without disturbing the limbs. (See next case.)

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* In describing specimens seen at the clinic of the late Professor Hoffa, Watkins (*Jour. A. M. A.*, November 20, 1915, 1803) states: "I noted also that the specimens from older children presented a ligamentum teres drawn out to the thinness of a thread, when it was not actually absent."

Dr. Murphy: I am happy that Dr. Porter has succeeded in making this bloodless reduction, as the operative reduction is a difficult and serious operation, but one that should always be resorted to when the bloodless operation is not advisable or fails.

Hibbs (*Jour. A. M. A.*, November 20, 1915, 1801) states: "A careful study of my failures in the treatment of congenital luxation of the hip has convinced me that some of those failures were due to anteversion deformity of the head and neck of the femur which was not recognized before operation. . . . I believe that this deformity of the femur is more often the cause of failure to secure ideal results (than defects in the acetabulum). . . . It would seem logical, therefore, to treat the matter as a twist of the shaft of the femur, and correct it by osteotomy (of the lower third of the femur) before any attempt is made to reduce the luxation."

Blanchard (*ibid.*, p. 1805), speaking of the structural changes in this luxation, states: "A shortened or twisted neck in a coxa valga position may look unpromising in the skiagram, and yet present no bar to a good reduction and ultimately to a good functioning joint. . . . If the skiagram shows delayed ossification from rickets or other causes, the reduction must be postponed.

"The best functional result may be obtained in children from three to five years of age and with from 2.5 to 4 cm. of shortening. . . . The difficulty of reduction increases with the shortening after 4 cm. have been passed. A reduction by steps can be gained in older children with 5 cm. or more of shortening by following the practice of the Vienna school.

"Reduction should be delayed in all cases of three years or younger and that show 2 cm. or less of shortening.

"Perfect or satisfactory function may be obtained in a large majority of the unilateral and a fair proportion of the bilateral cases."

CONGENITAL LUXATION OF HIP—BLOODLESS REDUCTION

Summary: Clinical history of patient; reduction; comments—mode of reduction; test of complete reduction; fixation by plaster-of-Paris.

CLINICAL HISTORY OF PATIENT

The patient, a female aged three years, was brought to hospital February 8, 1915. The family history is negative.

The child was born in a normal labor. The mother states that the child began to walk at the age of fourteen months, and that at that time there was no difficulty. There was a bulging of the abdomen, which the other children of the family did not show. In a short time the mother's attention was called to the fact that there was a deformity on the left side and a positive change in the character of the movement of the left lower extremity at the hip. Pain was absent. The mother noticed that, after sleeping or sitting for a time, upon getting up again the child favored the left hip in walking. After walking she often stumbles, falling usually forward upon the hands, but she straightens up immediately after the fall, raising the right side first and then pushing herself erect by pressing the hands upon the knees. There is no history of an injury.

OPERATION

Bloodless reduction was effected by Dr. Murphy's associate, Dr. Philip H. Kreuscher, on February 9, 1915.

COMMENTS

Dr. Murphy: Although this child is very stout, the extension that had been maintained for a week has had the effect of loosening up the muscles enough to justify an attempt at reduction. After stretching the attachments of the adductor group of muscles to the pelvis, the thigh was flexed upon the abdomen, and the leg upon the thigh. Then the head of the femur was pushed downward until it reached a point that was a little below the level of the acetabulum. Having reached that point, the head was rotated anteriorly by everting the limb, and could readily be seen slipping into the acetabular cavity. Then the limb was superlatively abducted. The transverse alinement was perfect. Both limbs were secured in the "frog" position by plaster-of-Paris (Fig. 307). The reduction was accomplished in less than a minute, but do not expect to make the reduction in that short time in many of your cases.

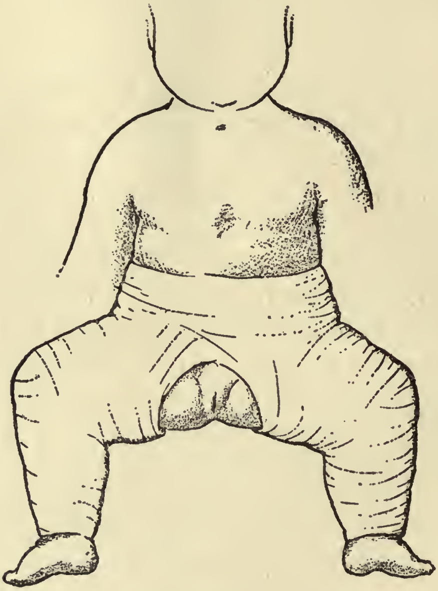

Fig. 307.—Sketch showing both limbs secured in the "frog" position by a plaster-of-Paris dressing. When one hip only is involved, it does not suffice to dress it alone in the extended and abducted position; both limbs must be placed in the same position, the better to control the pelvis.

When the reduction is complete, and with the limbs in this "frog" position, a line drawn from the internal condyle of one femur passes transversely across the lower border of the symphysis pubis to the internal condyle of the opposite femur (Fig. 308); that is, it passes through the heads of the femora. The legs are at right angles to this line, and the feet are on a line parallel to the one which passes between the femoral condyles; that is, the limbs are on a line that is parallel with the transverse diameter of the pelvis, and in a position of superlative external rotation (Fig. 308).

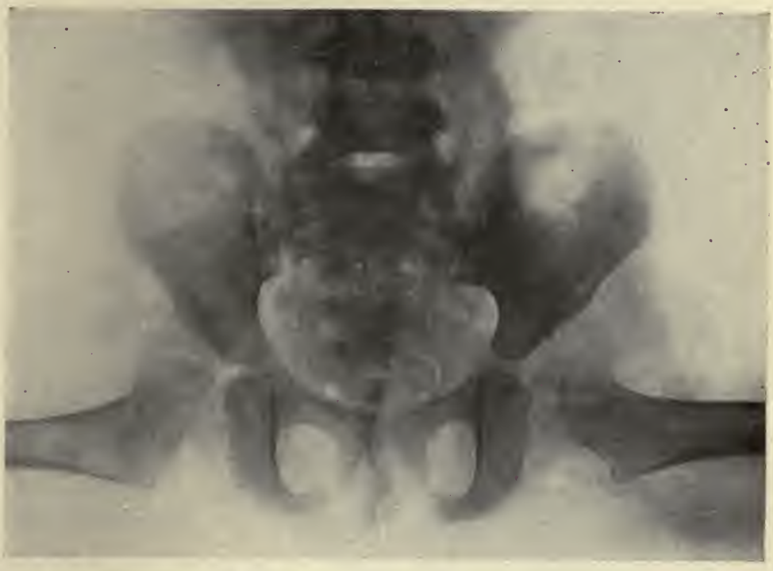

Fig. 308.—Skiagram taken after reduction of luxated left hip, showing restoration of head of left femur into acetabulum. Note shallowness and smoothness of acetabular rim on left side; compare with the rougher and more projecting acetabular rim on the right. Limbs in frog position (anteroposterior view).

On April 8, 1915, the plaster-of-Paris dressing was removed and a new one was applied.

(For a general discussion on this topic see the following case.)

[*N. B.*—This case progressed without untoward complications.]

CONGENITAL LUXATION OF BOTH HIPS —BLOODLESS REDUCTION

Summary: Clinical history of patient; comments—normal anatomy of hip-joint; mechanism by which head of femur is held in acetabular cavity; pathologic anatomy of congenital luxation of hip-joint; symptoms and diagnosis; treatment—reduction to be effected manually by the bloodless method and not by the use of traction devices; method employed by Dr. Murphy; position and mode of fixation after reduction; high percentage of good results from this method.

CLINICAL HISTORY OF PATIENT

THE patient, a male aged three and one-half years, was brought to Mercy Hospital October 22, 1915. The family history revealed no instance of congenital deformity or defect. The patient is the first and the only child of healthy parents. He has at no time been seriously ill.

When the child was two and one-half years of age the mother noticed for the first time the peculiar waddling gait, so characteristic of luxations of the hip. A skiagram revealed displacement of both femoral heads onto the dorsum of the ilium, to an extent of 2¼ inches above the normal site of the acetabulum. Upon closer scrutiny one finds two moderately developed acetabular cavities and two poorly developed femoral heads, the right head being placed more nearly opposite the middle of the neck, while the left is nearer the upper and outer aspect of the neck.

COMMENTS

DR. KREUSCHER (October 23, 1915): In the successful reduction of luxations of the hip-joint an accurate conception of the anatomy is essential. At the meeting of the ilium, the ischium, and the os pubis there is found the cup-shaped acetabular cavity in which the rounded head of the femur normally rests (Fig. 309). The mechanism by which the head is held in the acetabular cavity is as follows:

First, and most important, retention is afforded by the joint-capsule, which arises from the entire circumference of the acetabular rim, and inserts into the anterior intertrochanteric line in front (Fig. 310), and the neck of the femur above the posterior intertrochanteric line behind (Fig. 309); of functional importance are the thickened capsular bands termed the iliofemoral, or Y-shaped ligament of Bigelow (Fig. 310), the ischiofemoral, and the pubofemoral, and, to less extent, the weak ligamentum teres, a rounded cord which passes from the notch and transverse ligament of the acetabulum to the depression in the head of the femur (Fig. 311). Secondly, the support afforded by muscles, including the gluteus minimus, the iliacus, and the psoas magnus, which, through a portion of their fibers, serve as tensors

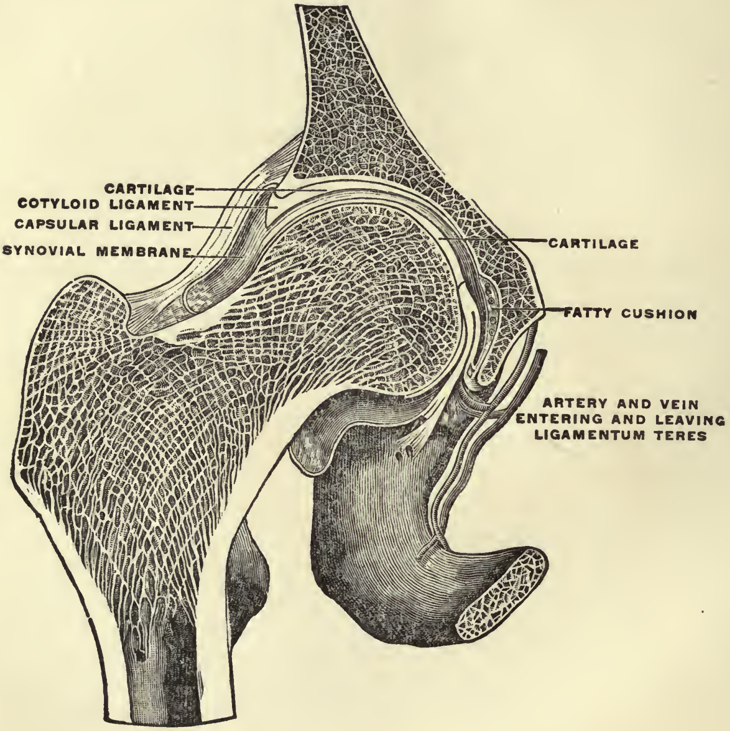

Fig. 309.—Hip-joint in coronal section (Gerrish, after Testut)

of the capsule; and, finally, the retention of the head of the femur by the depth of the acetabular constituent of the ball-and-socket joint (Fig. 309). Failing one or more of these essentials of the normal mechanism by which the integrity of the hip-joint is maintained, we have three varieties of congenital luxations, according to the direction the luxating head takes, namely, the upward and slightly backward, which is the most common; the posterior, and the anterior.

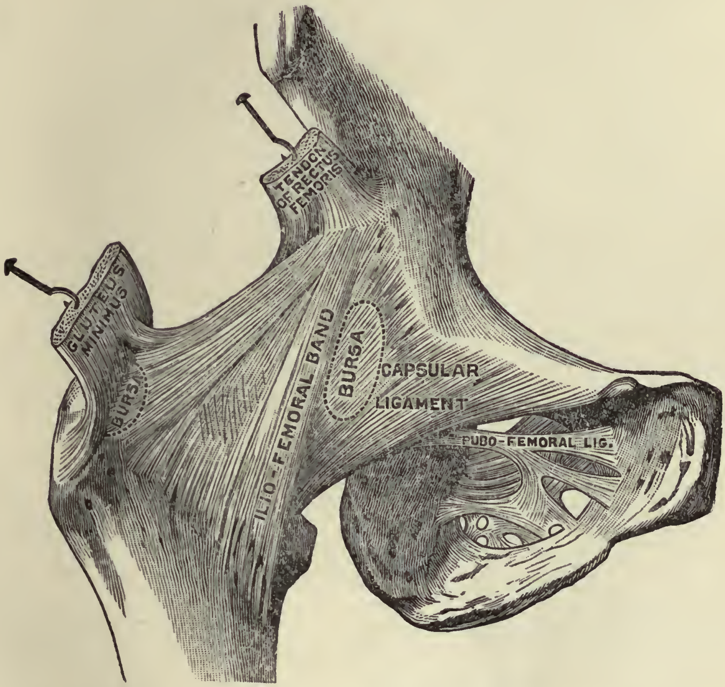

Fig. 310.—View of anterior capsule of hip-joint. Note strong iliofemoral band (Y-ligament of Bigelow) extending from anterior inferior spine of ilium (just below origin of straight head of rectus femoris) to anterior intertrochanteric line of femur. The two limbs of the Y and the less thickened portion of the capsule enclosed by them are distinctly shown. Note also the pubofemoral ligament (Gerrish, after Testut).

With regard to the pathologic anatomy of congenital luxation of the hip-joint several theories have been propounded. Hereditary influence is said to have been observed to play a part. Many surgeons still accept the mechanical theory of displacement; but we believe that Ridlon's radiographic demonstration at the last Atlantic City meeting of the American Medical Association proved conclusively that a congenital defect of the acetabulum existed both in the presence of luxation and in the absence of luxation, the degrees of stability of the hips varying from those that could not be luxated without first fracturing the femur, to those that could not be retained, no matter by what means, nor how many times the head was replaced.

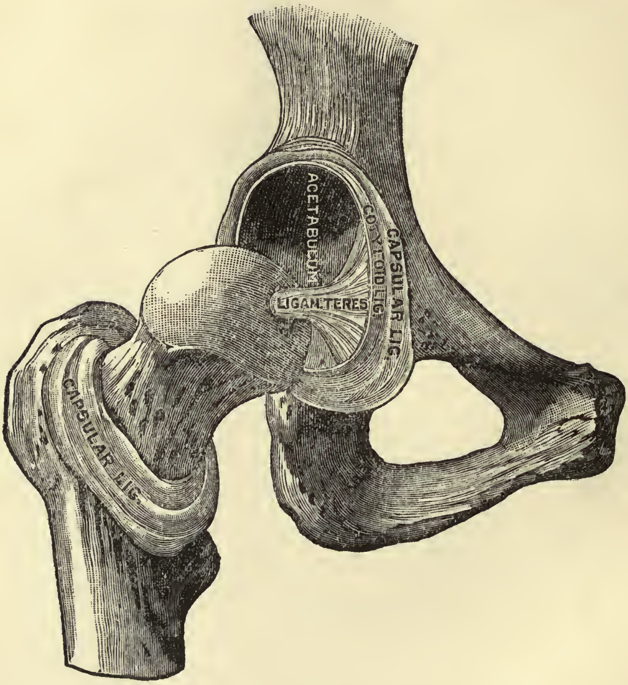

Fig. 311.—Hip-joint (front view). The capsular ligament has been largely removed (Gerrish, after Testut).

From radiographic studies of congenitally luxated hip-joints the following abnormities were noted. The acetabular cavity, although present in every case and normal in position, was always shallow (Fig. 307, p. 1171) and generally triangular in shape. The head of the femur rested above, above and behind, or anterior to the acetabulum.

SYMPTOMS AND SIGNS

At birth the displacement of the head upward is not particularly marked, but with weight-bearing this upward displacement becomes one of the most important features of the condition (Fig. 312). In unilateral cases asymmetry of the pelvis is liable to result during the progress of growth. Upon palpation, in the forward luxations, when the limb is rotated inward, the head can be felt before, but not behind. In the backward luxations, upon inward rotation of the limb, the head can be felt behind, but not before. In the upward luxations, on outward rotation of the limb, the head can be felt before, and on inward rotation, behind.

In double congenital luxation the children learn to walk late, and the gait is characterized by marked waddling or swaying from side to side. When the patient stands, there is marked lordosis, the perineum is broadened, and the trochanter and buttocks are prominent (Fig. 305, p. 1160). In the unilateral cases there is marked limping, and the abdomen seems prominent, especially on the affected side: in the previous case this sign was noted by the parents. The patients rarely complain of pain.

DIAGNOSIS

The diagnosis is made from correlation of the physical and skiagraphic findings. The waddling gait is almost pathognomonic. In cases of upward or upward and backward luxation the great trochanter lies above Nélaton's line on the affected side, and during manipulation the head can be felt to slip about. Abduction of the limb is limited in extent. Symptoms and signs of hip-joint disease are usually absent. In a large majority of cases the skiagram will reveal the condition of luxation (Fig. 306, p. 1162).

TREATMENT

Since the early and successful manipulative reduction of Ridlon, Whitman, Bradford, Lorenz, and others, treatment by the use of apparatus no longer holds a place. In effecting reduction the various traction devices, including special tables for traction, should be used rarely, if at all. A congenitally luxated hip that can be safely reduced upon a special table, by a mechanical device or a cutting operation, can be reduced equally well manually upon a sand-bag by a surgeon of ordinary physical strength, who knows how to manipulate the limb properly.

The two methods of effecting manual reduction of the displaced head are, first, by manipulation without incision (the *bloodless* method); secondly, by incision and manipulation (the *bloody* method). In the large majority of cases the bloodless method is the one of choice, and if, after repeated attempts, it should fail, the surgeon is justified in using the bloody method. The old "bloodless" method of Lorenz, in which the adductor muscles were torn or lacerated, often resulted in paralysis of the limb, fracture of the femur, or both, and therefore should be abandoned, for one should remember that, after reduction has been effected, the adductor muscles are of great value in retaining the head in the acetabular cavity.

In effecting manual reduction we avoid the frequent occurrence of paralysis and the occasional rupture of the perineum by not stretching the limb, as Lorenz did, by a downward pull; we do not try to stretch the joint-capsule by flexing, hyperextending, and abducting the limb while extended at the knee, nor do we abduct the thigh beyond the position of 90 degrees of flexion, nor deliberately tear the adductor muscles by hacking at them with the ulnar border of the hand. By omitting such undesirable manipulations we avoid much subcutaneous hemorrhage and the subsequent formation of a hematoma, which may need aspiration, and even incision and drainage. We do not approve, either, of the use of Koenig's wedge-shaped block placed behind the joint as a fulcrum, since in all cases this procedure results in bruising of the tissues, and in some, in fracture of the neck of the femur. The method of choice is one by which the reduction can be effected with the least degree of trauma and minimum shock to the patient. Such a method is employed in this Clinic, and is carried out as follows:

THE MURPHY CLINIC METHOD OF REDUCTION

In the first instance, before attempting reduction in strong, robust children beyond three and one-half years of age, where the adductor muscles are tense, Buck's extension apparatus with traction of from five to eight pounds—the weight varying with the age and muscular development of the patient—is applied with the limb in abduction, and retained several weeks. If necessary, the Rainey splint, which has been previously described and illustrated in the CLINICS (April, 1912, p. 173), is used, for it results in perfect maintenance of abduction. By these simple methods the muscles and tendons are gradually, but surely, stretched, and with very little pain, or none at all, and without causing subcutaneous hemorrhage. In case the adductors are not tense, this portion of the treatment may be omitted, and then we proceed as follows:

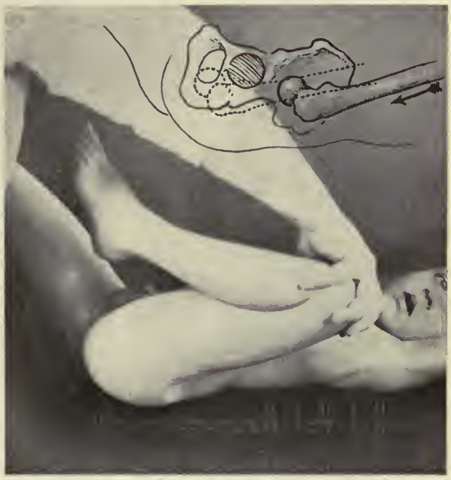

Fig. 312.—Reduction of luxated hip by bloodless method. Left hip involved. First step in reduction. Knee grasped with operator's right hand, and limb is fully flexed at both hip and knee. Pressure made directly downward upon knee, so that head of femur is driven backward and downward below level of acetabulum. Outline-insert shows excursion of head of femur during manipulations; dotted line shows position of femur after this movement is completed (lateral view).

The patient is placed upon his back on the table, and is deeply anesthetized. In cases of long duration the hips are elevated upon a sand-bag or folded towel placed beneath the pelvis. In reducing a left-sided luxation, for example, the knee is grasped with the operator's right hand, and the limb is fully flexed at both the hip and the knee (Fig. 312). The knee points upward toward the axilla, and the head of the femur is directed downward. Pressure—as much as seems necessary—is now made directly downward upon the knee, so that the head of the femur is driven backward and downward below the level of the acetabulum. Counterpressure is made upon the tuberosity of the ischium, and the position of the femoral head is determined by the fingers of the left hand, the thumb of which rests in front, below the groin,—that is, in front of the acetabulum,—while the fingers are situated behind, grasping the head and neck and the great trochanter of the femur (Fig. 313). Slight outward traction upon the knee is necessary in order to stretch the adductor tendons. If, by pushing the head downward below the acetabulum, it can be brought low enough to effect reduction, slight inward rotation of the thigh is made; and after the head has thus been brought behind the level of the acetabulum, the thigh is slowly abducted, and the head now passes upward over the lowest portion of the acetabular rim into the acetabular cavity (Fig. 314). These manipulations can often be effected very quickly, and are easy if the surgeon has a clear mental picture of the orientation and movements of the head during the time of his manipulations (Figs. 312, 313, 314). "In reducing luxations of the hip it may be noted that the internal condyle of the femur faces in nearly the same direction as the head of the bone" (Treves).

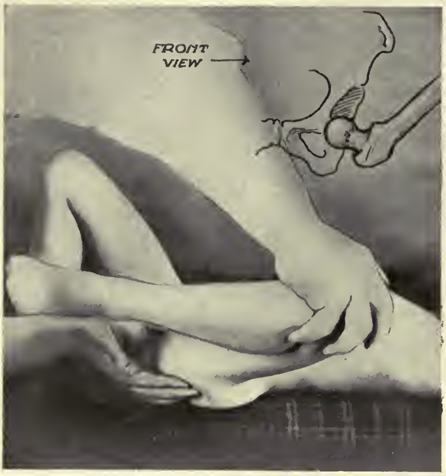

Fig. 313.—Outward traction upon knee made to stretch adductor tendons. Shows counterpressure being made upon tuberosity of ischium with fingers of left hand, thumb of which rests in front of acetabulum and below groin. Fingers of left hand also determine position of femoral head. Outline-insert shows head resting below acetabular margin, as at end of first step.

TREATMENT AFTER REDUCTION

After reduction the limb is left remaining in the position of right-angled abduction with nearly the same degree of outward rotation (Fig. 308, p. 1171). A gypsum case is then applied snugly, after the bony prominences have been carefully padded with sheet-wadding, and includes the pelvis and the limb down to the ankle (Fig. 307, p. 1170). A gypsum case that is applied down to the knee only is objectionable in that it does not prevent the limb from rotating. Fixation by the original gypsum case is maintained for from eight to twelve weeks, and then, with the knee several inches lower and the limb in a position of slight inward rotation, a second gypsum case is applied.

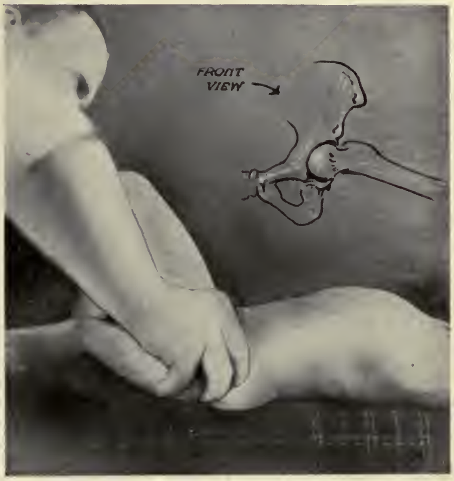

Fig. 314.—Final step in reduction. After slight inward rotation of thigh the latter is slowly abducted, and the head now passes upward over lowest portion of acetabular rim into acetabular cavity. Outline-insert shows head "at home."

Upon the whole, the limb should be immobilized by plaster for a period of from eight to twelve months, the time varying with the character of the case.

After this method of bloodless reduction perfect anatomic and functional results have been estimated by various operators as having been obtained in from 50 to 78 per cent. of the cases.

OLD FRACTURE-LUXATION OF RIGHT HIP-JOINT—REDUCTION AFTER EXCISION OF HEAD OF FEMUR

Summary: Clinical history of patient; comments—significance of the relations of the anterior superior iliac spines to the long axes of the limbs; operation—exposure—pathologic anatomy described—landmarks of hip-joint obscured; detached fragment of head removed from acetabulum; resection of neck of femur to extent necessary to effect reduction; neck of femur placed in acetabulum; closure of wound; recapitulation; postoperative comments.

CLINICAL HISTORY OF PATIENT

The patient, a male aged thirty-four years, was admitted to hospital September 10, 1914. The family and past histories are negative.

The present condition is the result of an injury received six months ago, in March, 1914, when the patient jumped in front of a switching engine and lost both legs—cut off in the lower third: this required operation for neuromata, which operation was previously published in the Clinics (October, 1915, pp. 1007–1011)—and sustained an injury to the right hip. He was taken to a hospital half an hour after the accident. The right hip was very much swollen, and a skiagram showed that the head of the femur was displaced upon the dorsum of the ilium (Fig. 315). All attempts at reduction proved futile.

At the present time there is motion in both hips—preternatural mobility on the right side. There is a bulging on the right side, between the posterior superior iliac spine and the ischial tuberosity. With the patient on his back, both anterior superior iliac spines are on the same level, but the right thigh lies across the midline of the left thigh.

COMMENTS

Dr. Murphy (September 12, 1914): With both the anterior superior iliac spines on the same level, the right thigh lies across the middle of the left thigh (Fig. 317). This is the feature I want to emphasize, for it tells the whole story of the situation before us. Owing to the shortening, in attempts to bring the right thigh parallel with the left, the right side of the pelvis, measured at the anterior superior iliac spine, ascends four inches. With the right thigh crossed over the left in this manner, however, the two spines are situated upon the same level. The patient's great difficulty is that both legs are partially off, as shown in Fig. 317, *A*, but a greater difficulty is that he cannot walk on artificial limbs, because the right thigh interferes with the left by crossing it. He is unable to use both artificial limbs and

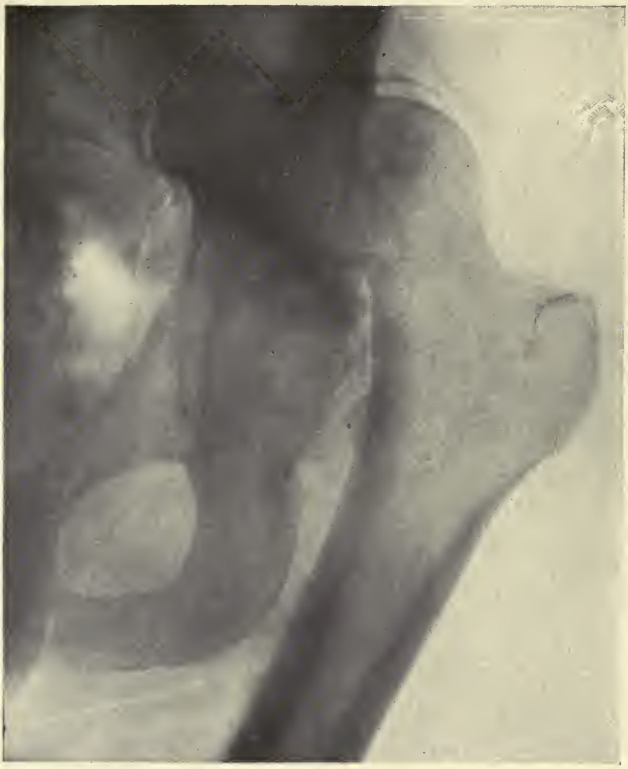

Fig. 315.—Preoperative skiagram of fracture-luxation of femur. Caput on dorsum ilii. Fragment of head in acetabular cavity (posteroanterior view).

crutches: if he had but one sound leg, he could manage to get around with the help of crutches.

Look at him now! You can see the deformity without my describing it to you, but the talk is for the record. The right anterior superior iliac spine is two inches higher than the left.

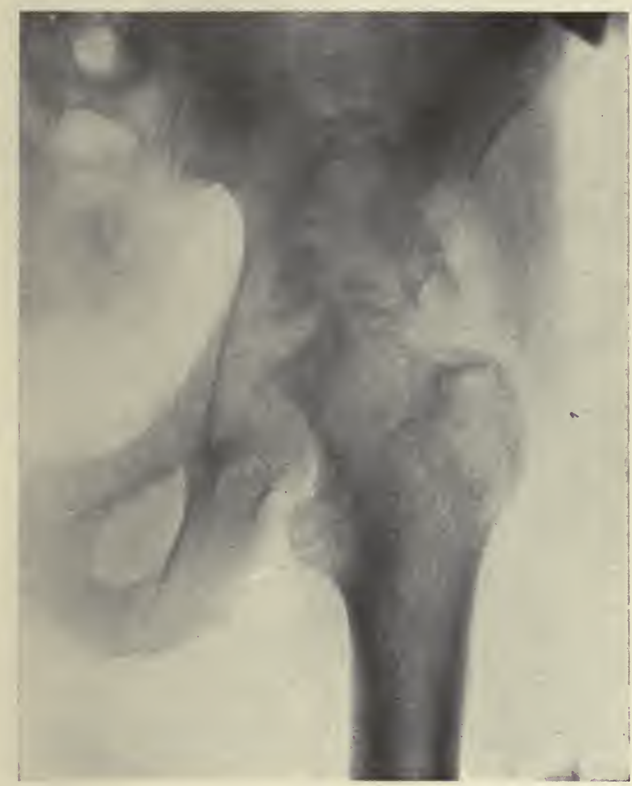

Fig. 316.—Postoperative skiagram showing reduction of luxation of femur by excising head of femur and placing cervical stump in acetabulum (posteroanterior view).

When the anterior superior iliac spines are on a plane that is parallel with the ground, his thighs are found crossed: that is the deformity to be overcome. Even though we have to excise the head of the femur to overcome the adduction, yet we must correct the deformity in such manner that this man will be able to walk.

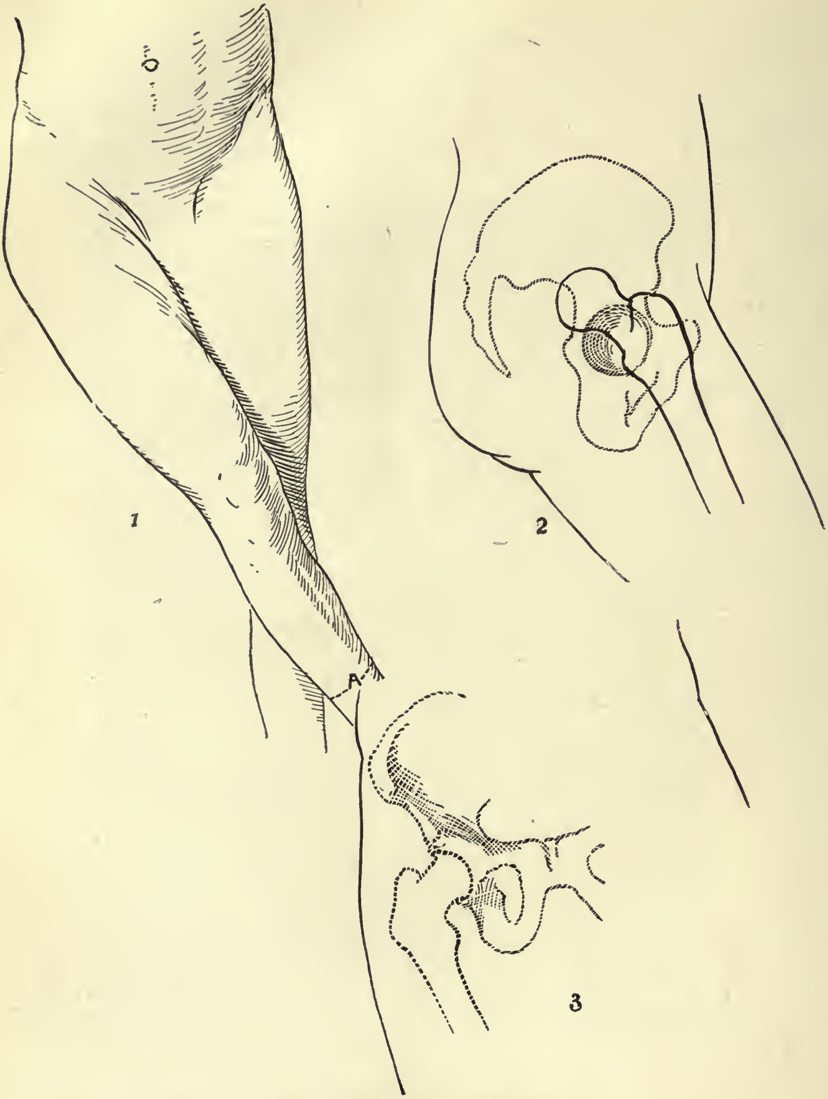

Fig. 317.—Sketches of luxation of hip, made at operating-table: 1, Shows degree of adduction present with upward and backward luxation of head of femur. Both legs had been amputated at point *A*. 2, Shows head of femur resting upon dorsum ilii just before reduction. (The detached fragment of the head of the acetabulum is not shown in this sketch.) 3, Shows head of femur amputated and cervical stump inserted into acetabulum. It was impossible to effect the reduction even by the open method, without excising the head.

We shall endeavor to accomplish reduction with the open operation.

The reduction of an ancient luxation of the hip is a hard proposition. Mechanical obstacles are hard to overcome, not to mention the risk of a cutting operation. With the latter the mechanical work is even harder—almost severe. The head of the femur will not go down in the direction of the acetabulum: therein lies the difficulty.

There is another feature in this skiagram to which I wish to call your attention. The skiagram gives the impression that a part of the head of the femur has been detached and still remains in the acetabulum (Fig. 315). Even though that be the real situation, it will not interfere with reduction of the luxation.

OPERATION

You can see that abduction is practically impossible. [Incision.] I am reflecting the gluteus maximus muscle to aid me in this work when I have freed the head: that muscle exerts powerful traction upon the femur from behind. The great degree of limitation of motion shows how much cicatricial fixation there must be; the adductor group is under great tension. I shall not divide this adductor group of muscles until after I have freed the head.

There is the head of the femur, and posteriorly the great sciatic nerve. There is the Y-ligament of Bigelow, passing across the front of the femoral neck. I am now dividing the pyriformis and the obturator and gemelli muscles or their tendons to expose the capsular ligament of the hip-joint, which is just beneath them. These muscles come forward from behind and are attached at or close to the tip of the great trochanter. There is the capsule of the hip-joint. I shall now try to find the acetabulum. Part of the head of the femur is separated, adding to the difficulty of developing the acetabulum in the operative field. That is the upper margin of the acetabulum: it is surrounded by a great cicatricial mass. Now I have exposed some landmarks for the first time. The first landmark indicates the position of the acetabulum, and in it is the detached fragment of the femoral head. The fragment has united to the acetabulum, and I must free it by removing its enveloping mass of fibrous tissue. We shall try to find another landmark—the upper margin of the capsule—by rotating the head. There is the detached portion of the head that was left in the acetabulum, as we had calculated from examination of the first skiagram. We were not sure of its presence, however, for, as you see, it is not large enough to cast a definite shadow upon the plate.

The acetabulum is now clear and distinct in the depths of the wound. I shall try to bring the head of the femur down by using levers, and replace it in the acetabulum; if I cannot do that, I shall have to excise the head. . . I have been able to drag it down to within an inch and a half of the acetabular margin but no farther: therefore, I shall now proceed to remove the head. The same difficulty is encountered when reducing congenitally luxated hips in adults. [The head of the femur was excised and the cervical stump placed in the acetabulum (Figs. 316 and 317, *3*).]

We do not foresee any probability of a recurrence of the luxation, although it may occur. I am putting my forceps upon the pyriformis and the obturator muscles. I am not reattaching them to the tip of the great trochanter, but along its edge, so that they will still retain some degree of function. The stump of the limb will be dressed and retained in abduction.

Notwithstanding all that hard work I think that we succeeded in keeping our hands out of the operative field the whole time, though it was a difficult proposition.

RECAPITULATION

Let the record show that there had been a fracture of the head of the femur and a fragment consisting of about two-fifths of the head rested in the acetabulum, the remaining intact portion being luxated upon the dorsum ilii (Fig. 315); that the head was easily exposed and the neck readily freed; that it was easy to remove the fragment of the head from the acetabulum; and that then the real difficulty began when we made several ineffectual efforts to reduce the head into the acetabulum. We then decided that the trauma of continuing such attempts would be too great, so that we finally excised the head, in pursuance of the plan which we had propounded before beginning the operation, when we said that it might be necessary to resect the head, and finally placed the cervical stump in the acetabulum (Fig. 317, *3*).

POSTOPERATIVE COMMENTS

I have ten or twelve cases with this adduction deformity with more or less destruction of the head waiting to come into hospital: from the difficulties seen in this operation you do not wonder why I keep them waiting. The chief difficulty lies in overcoming the long-standing and vicious contracture of the adductor muscles.

[Cases of fracture of the neck of the femur have been published previously in the CLINICS for April, 1912, pp. 165–175 (illustrations of Rainey splint on p. 173); February, 1913, pp. 16–27 (five cases with eight illustrations); June, 1913, pp. 421–429 (displacement of head upon dorsum ilii).]

[*Note.*—The case progressed without untoward complications.]

COXA VARA (BILATERAL) DUE TO STATUS LYMPHATICUS HYPERTHYMICUS—PROGRESS UNDER CONSERVATIVE MEASURES

Summary: Clinical history of patient; examination; treatment; progress of case; recapitulation; diagnosis from tuberculous coxitis; marked improvement under conservative measures; *status lymphaticus hyperthymicus.*

CLINICAL HISTORY OF PATIENT

The patient, a male aged fifteen years, who had always been an extremely fat boy, came to the office on September 1, 1914.

Over two years ago, on May 17, 1912, he fell a distance of 18 inches and landed upon the left hip. The injury caused severe pain. Physical examination revealed eversion of the left foot, but no shortening: there was also crepitation. A gypsum case was applied and was renewed four days later, on May 21, 1912, and again at the end of six weeks.

Four months after the accident, on September 17, 1912, the gypsum case was removed and crutches were substituted, and the patient was permitted gradually to bear weight upon the left lower limb, while in November, 1912, he discarded the crutches and walked without pain.

In the latter part of December, 1912, seven months after the fall, a wagon-wheel ran over the right foot, breaking down the arch. For this injury a gypsum case was applied and retained several weeks.

Examination of the left hip at the end of the first year, and again at the end of the second year, after the fall, revealed a wide range of flexion and of extension; but within the last six months limitation of motion of the joint was manifested, and has increased gradually.

Six weeks ago, on July 15, 1914, the boy noticed that, after sitting down for a while, upon arising to walk the right hip had become stiff, and that, upon walking, motion was painful at first, but the pain gradually diminished as he went about: he was not, however, incapacitated. This state of affairs continued two weeks.

There is a history of exposure in that the patient had been in swimming for a long time every day for two or three weeks.

One month ago, on August 1, the pain in the left hip became so acute that the patient had to take to bed, and was bedridden two or three days. There were no signs of bacterial infection at this time, and, aside from the pain, he was not incapacitated. Two weeks later, on August 15, he went away to a lake region, where he again indulged in swimming. One day after an uphill walk he again became suddenly incapacitated: no signs of infection were present this time either.

The tonsils and adenoids were removed about three years ago. The patient has no evident thyroid enlargement, nor has he a palpable thyroid. He has always been very stout, now weighing, at the age of fifteen years, 145 pounds.

EXAMINATION

Dr. Murphy (September 4, 1914): Examination shows a double coxa vara, which is severer on the right side than on the left. Skiagram shows in each case a bowing down of the head and neck of the femur (Figs. 318 and 319). The softening of the cartilaginous epiphyseal line permits the bony epiphysis to rotate on the neck, and leaves a gap superiorly. The thymus gland is not palpable, nor is the thyroid. The transverse process of every cervical vertebra can be palpated, showing how free the neck is from swellings.

The patient presents the typical picture of a case of *status lymphaticus hyperthymicus.*

He has the classic waddling gait of a double coxa vara. It somewhat resembles the gait of a double congenital hip-luxation, but there is absence of posterior prominence of the trochanters, and pronounced lateral prominence, as is typical for these cases.

TREATMENT

Each limb will be dressed in the position of abduction, and to each will be applied double extension with the traction of a 20-pound weight. The patient is to take iodothyrin and desiccated thyroids. This treatment will be continued three months.

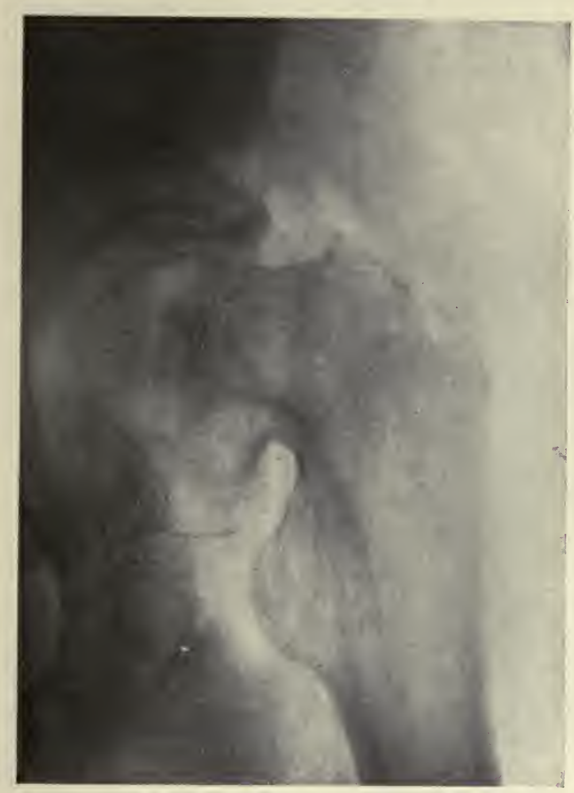

Fig. 318.—Skiagram of right hip showing coxa vara. Note great deformity of cervix and caput femoris. Before treatment.

PROGRESS OF CASE

November 3, 1914 (at the end of nine weeks' treatment): Skiagram reveals extraordinary improvement.

December 2, 1914 (at the end of thirteen weeks' treatment): Judging from the perfect definition of the head and of the neck that is revealed by skiagrams, the osteoporotic process has been checked and there is an eburnating process going on now in the head and neck of each femur. All that is necessary to prevent reëstablishment of the process at this stage is the *gradual resumption of activity.* His walk will be wobbly on account of the approximately 90 degree relationship which each neck forms with the shaft of the femur, instead of the norma of 130 degrees.

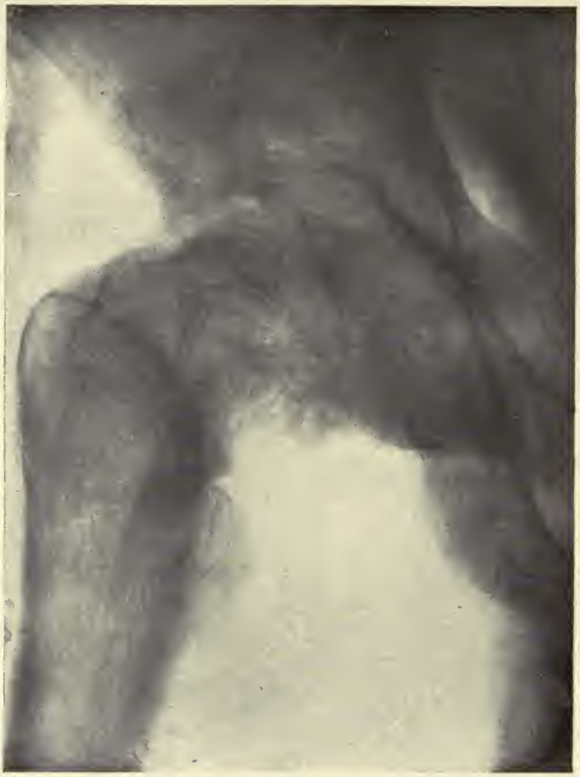

Fig. 319.—Skiagram of left hip, showing coxa vara with great deformity of caput and cervix femoris. Before treatment.

The weights are to be taken off and left off in the daytime, first for one-half hour and then for one hour, daily increasing the intervals at that ratio and gradually eliminating the weights until, by the end of two weeks, the boy is able to be about on his feet for three hours at a time; and he is to be sitting up between times.

December 24, 1914 (at the end of sixteen weeks' treatment): The measurements of both limbs are about equal. On the right side there is no longer any limitation of motion. The boy seems to be getting well. He may now get about with the aid of crutches. He is to resume free use of the limbs gradually, but never to the extent of causing pain.

January 26, 1915 (at the end of twenty-one weeks' treatment): The boy weighs 134 pounds—a decrease of 11 pounds since the beginning of treatment, five months ago: the bones are becoming solid. He is still using crutches, and is now able to go back to school.

RECAPITULATION

Dr. Murphy (January 28, 1915): Just this week we had in the office a boy who came in about five months ago complaining of pain in the right hip. Judging from the appearance of the limb alone the condition resembled early tuberculosis of the hip-joint, but upon noticing the amount of adipose tissue over the whole body we recognized at once that we were not dealing with a case of tuberculous coxitis, but with one of coxa vara, and that the angle between the neck and the shaft of the femur was gradually deviating from the norma of 130 degrees to a right-angle relationship, thus increasing the risk of fracture. Within a few weeks the same process began in the left hip-joint. We ordered for him iodothyrin and desiccated thyroids, put him to bed for a period of three months, and applied Buck's extension to both limbs. These cases of oversecretion of the thymus gland respond very well to the thyroid gland treatment.

The boy was again in the office yesterday, and on weighing him we found that he had lost eleven pounds, so that he now has about the average weight for a boy of his age. In order to prevent these thigh bones from turning in we are still having him use crutches. At the present time the head and neck of each femur show compact bony tissue in the skiagrams.

[*Status lymphaticus hyperthymicus* was referred to in the

CLINICS for August, 1915, p. 700, in connection with a case-report of metastatic thymus tumor in the breast.]

The treatment of all these cases should be a prophylactic one. These hyperthymus cases are so easily diagnosed before the osseous changes take place that he who runs may read and the proud father and mother of these superlatively fat boys and girls should be informed that this fat is a pathologic condition that should and can be remedied by proper treatment, thereby avoiding the later crippling lesions of the hips. The iodothyrin seems to be as advantageous in these cases as the desiccated or fresh sheep's thyroid. This patient was last seen on July 2, 1915. He is in splendid condition, although greatly reduced in weight.

I. RECENT COMMINUTED T-FRACTURE IN LOWER THIRD OF FEMUR—OPERATIVE REDUCTION—PLATING

II. RECENT FRACTURE IN LOWER THIRD OF FEMUR—OPERATIVE REDUCTION—PLATING

Summary: Clinical history of patient; comments—nature of injury and of deformity; danger of fragment lacerating popliteal vessels; two other dangers associated with fracture at this site; operation—importance of no-hand-contacting technic; anxiety caused by these cases; vicious action of gastrocnemius; muscle found interposed between bone-ends; lower fragment comminuted; periosteum removed from bone-ends to favor union and to prevent excessive callus-formation; fragments levered into place and maintained by Lane plate; postoperative comments—importance of an ample incision; coffer-damming of tissues; plaster dressing—use of Gigli saw—protection of peroneal nerve from pressure. Additional case—plating of a fracture of femur.

CLINICAL HISTORY OF PATIENT

The patient, a female aged thirty-four years, was admitted to hospital January 16, 1915. The family history was negative. She had had the usual diseases of childhood. Six years ago, in 1909, the left radius and ulna were fractured: one month after the injury the forearm was operated upon and the bones were wired.

The present condition is the result of an injury that was received yesterday—on January 15, 1915. While getting out of a window on to a sleeping-porch the ledge of the porch gave way and the patient fell 20 feet to the ground. Examination revealed fracture of the lower third of the shaft of the right femur, simple in type. This clinical diagnosis was confirmed by skiagrams and, in addition, a comminuted fracture of the lower fragment was revealed (Fig. 320).

Five hours after the accident the patient was removed to this hospital and Buck's extension apparatus was applied to the limb.

COMMENTS

Dr. Murphy (January 30, 1915): The patient is a very stout woman, weighing more than 200 pounds. This is what happened to her two weeks ago. She wished to go from a sleeping-porch on the second floor of her house to her room, but discovered that she had unintentionally locked the door behind her. Then she attempted, as she had succeeded in doing so many

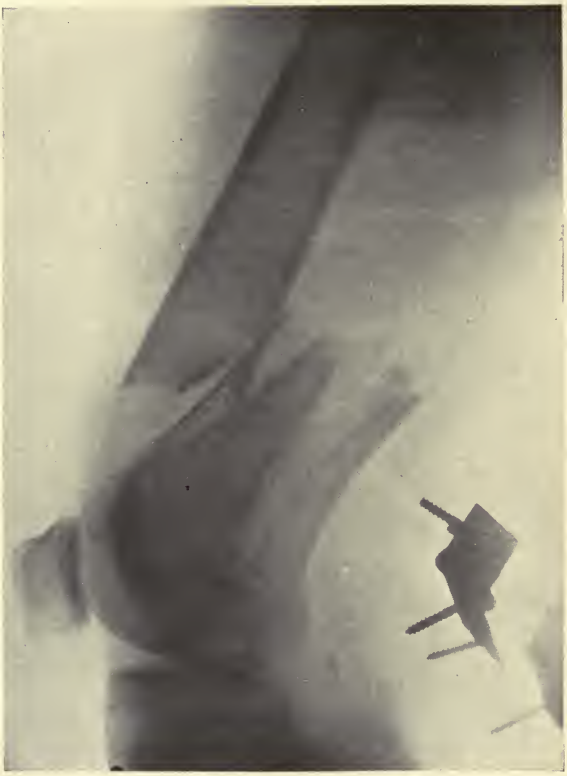

Fig. 320.—Preoperative skiagram showing comminuted fracture of lower third of femur, with tilting of upper ends of lower fragments backward by the contraction of the gastrocnemius muscle, endangering the popliteal vessels (lateral view).

times before in the summertime, to crawl along an adjoining ledge to reach the window of her room. The ledge was coated with ice, and she slipped and fell to the ground, a distance of 20 feet, fracturing the right femur in the lower third of the shaft. Examination revealed displacement of the distal fragments posteriorly (Fig. 320), and overriding of the proximal fragment to such an extent that it seemed to be under the patella, and that union would be impossible: even with the patient under anesthesia we could not reduce the fragment. Then we postponed operative treatment two weeks. There was an enormous effusion in the knee-joint and a hematoma about the site of fracture. At the present time the effusion has almost all been absorbed, and the hematoma has practically disappeared. Now we have to deal with the proposition of effecting reduction and maintaining it.

Given a fracture of the lower third of the shaft of the femur *above* the origin of the gastrocnemius attachment, one is haunted by the possibility of a very dangerous situation, namely, that the popliteal vessels have been lacerated by the backward displacement of the upper end of the lower fragment. If this calamity be present here, we shall have to ligate the femoral artery high up. Recall the patient who suffered hemorrhage from the popliteal artery: he was so exsanguinated that we were compelled to transfuse him upon the table in order to save his life.

There is a triple danger present when one deals with fracture of the lower third of the shaft of the femur. The first danger arises when the gastrocnemius muscle by its contractions flexes and pulls the bone with its sharp spicula back against the popliteal vessels. The second danger is associated with the difficulty of securing the distal fragment and retaining it in position so that it will not drop back again after reduction. The third danger—a colossal one—is that of splitting of the lower fragment into the joint, thus admitting of a direct arthritic infection, for if the danger were realized, we would have to deal with a compound comminuted fracture complicated by an opening into the knee-joint, which might result in (1) necessity of amputating the limb; (2) the danger of death from sepsis, both before and after the amputation. No case that one would encounter within a year's time could threaten the patient with a graver danger than that with which a fracture in this situation can.

OPERATION

[Makes a large incision.] This work must be performed without hand-contacting, otherwise it had better be left undone. This no-hand-contacting technic puts the operator under tension, because we are so accustomed to using our hands in abdominal work. If in bone work you let the glove or the hand get into the wound, it is only a question of time until the percentage of infections begins to rise. You see how desirable it would be to use your fingers in dealing with these displaced bone-ends. It will be our endeavor to maintain the reduction by passing long screws across the femur from the external to the internal condyle. We do not expect to hold the fragments absolutely fixed by the screws. There is no remuneration that compensates for the anxiety that one expends over these cases. Twenty-five cases of appendicitis, good, bad, and indifferent in type, are not equal to the mental strain of operating upon one of these fractures.

There you see the patella resting on the lower end of the upper fragment. It is the gastrocnemius muscle that is responsible for the mischief. When we have a fracture of the femur in children above the lower epiphysis, we overcome the displacing action of the gastrocnemius by merely flexing the leg acutely upon the thigh—in effect, by making a splint of the leg to retain the fractured ends in apposition. I cannot approach the fragments by an anterior incision through the rectus muscle as I should like to, for if I injure the tendon of the quadriceps extensor muscle, in healing it will become adherent to the periosteum, and the patient will be crippled through loss of extension power. There is the vastus internus muscle: a portion of it intervenes between the ends of the bones, and would have prevented union of the fragments. Now I have freed the interposed muscle and I shall remove it.

Let the record show that the lower fragment is the more comminuted.

You can see the split in the lower fragment (Fig. 321). [Applies levers to the displaced fragments.] Now we have secured both fragments in good apposition and good alinement. In performing these operations we always remove the periosteum from the end of each fragment, thereby affording a better opportunity for union and preventing excessive callus-formation on the bone-ends. When I called attention to the rôle of the periosteum in transplants I said that although we do not consider the periosteum essential, yet we believe that it is osteoconductive.

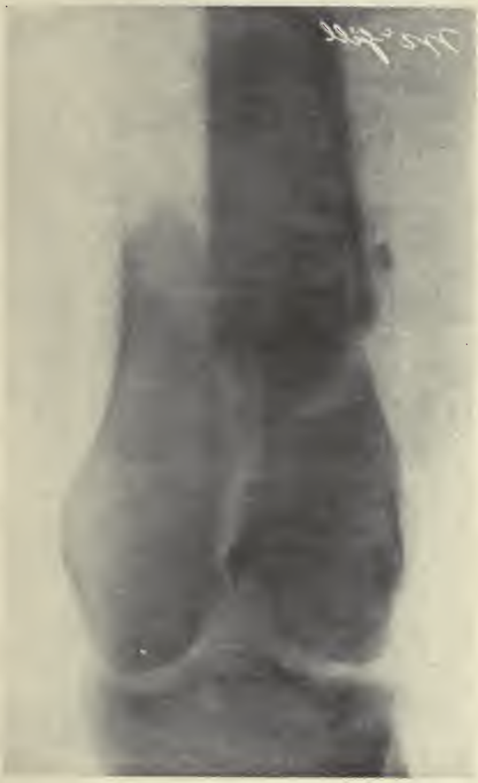

Fig. 321.—Preoperative skiagram showing T-fracture of lower extremity of femur (anteroposterior view).

. . . [Inserts Lane plate: uses long screws to produce fixation of the split lower fragment (Figs. 322 and 323).] We suture the edges of the fascia lata firmly together to avoid hernia of the quadriceps muscle. [Sutures superficial fascia and skin.]

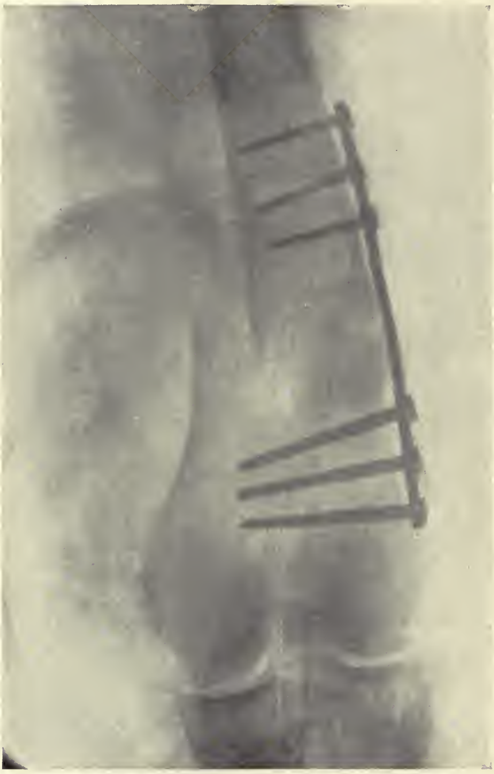

Fig. 322.—Postoperative skiagram of comminuted T-fracture of lower third of femur. An eight-screw Lane plate was used; three screws were inserted into the upper fragment and three into the lower fragments, the central screw-holes falling too close to the transverse plane of fracture to take screws. Note that the lower three screws are extra long, reaching across so as to secure the fragment on the inner side (anteroposterior view).

RECAPITULATION

Let the record show that we succeeded in bringing the retroposed fragment back into position with the aid of the skid; that the fracture was plated and secured in position without hand-contacting; that an eight-screw plate was inserted, three screws securing the upper fragment and three the lower, the middle screw-holes falling directly over the comminuted part so that screws could not be inserted through the central holes (Fig. 323); and finally that the wound was closed with horsehair, but not sealed with collodion.

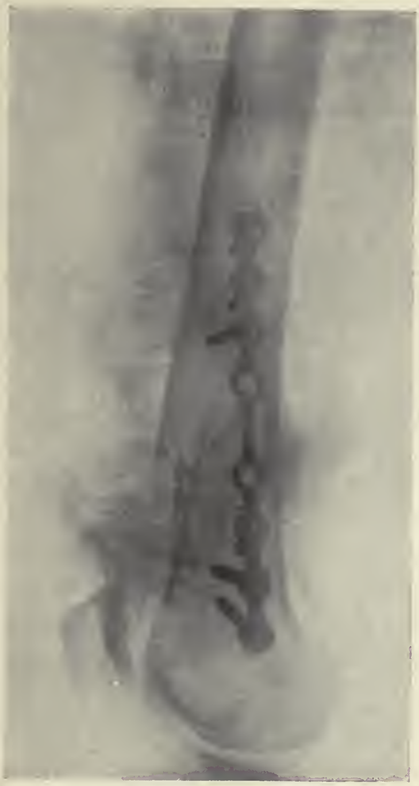

Fig. 323.—Same as previous figure (lateral view). To appreciate extent of reduction compare with Fig. 320, p. 1198.

POSTOPERATIVE COMMENTS

In operating upon fractures the very first step is to make an ample incision, so as to obviate the necessity of subsequently enlarging the wound to adjust the fragments. This work is not completed until the patient is back in bed with the fragments still in good position.

We are operating two weeks after the fracture occurred, so that the tissues have had time to become coffer-dammed.

Dressing.—[Applies plaster-of-Paris bandages.] We have placed the Gigli saws beneath that plaster case to facilitate its removal. Had we placed but one saw beneath the dressing, upon removal of the latter we might saw into the patella, owing to the anterior angulation that forms at that point. This use of the Gigli saw was suggested by Dr. Bechtold, of Indiana, a former intern of mine. It is a wonder that this method had not been thought of long ago, as a substitute for our nerve-racking and patience-taxing methods of cutting gypsum cases, which we employed so many years.

We do not have to protect the external popliteal nerve, because we covered it over with the big dressing for the wound. The external popliteal nerve is the one nerve in the lower extremity that suffers from pressure paralysis, because of its exposed position as it winds around the neck of the fibula just below the head. This nerve is exposed to pressure from both a Buck's extension apparatus and a plaster-of-Paris dressing, and pressure results in drop-foot unless it be detected early and is relieved.

[*Note.*—There was complete primary union, both of the soft parts and of the bone. A perfect result has been obtained with flexion to a little better than right angle at the present time. The patient will eventually have freer flexion and extension.]

II

RECENT FRACTURE OF SHAFT OF FEMUR—OPERATIVE REDUCTION—PLATING

Summary: Clinical history of patient; comments and operation; recapitulation; postoperative comments.

CLINICAL HISTORY OF PATIENT

THE patient, a male aged eight years, was admitted to hospital October 19, 1914. The family history was negative. Six months ago he had pertussis.

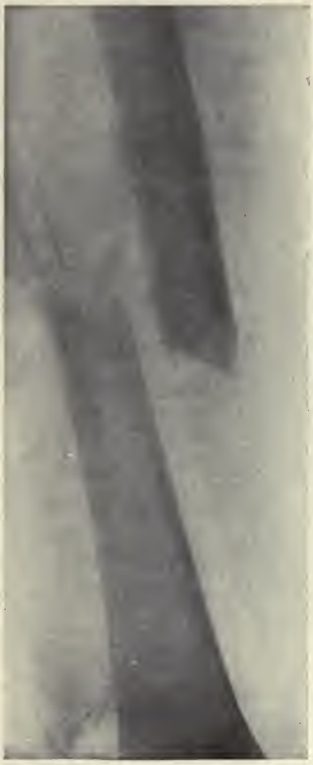

Fig. 324.—Preoperative skiagram of fracture of shaft of femur, showing extent of displacement of fragments (anteroposterior view).

The present injury was received the day of admission. While crossing the street the patient was struck by a moving automobile and knocked to the ground. He tried to get up, but could not on account of the severe pain in the left lower extremity above the knee. No bones protruded through the skin. The foot was everted. The patient was brought to hospital and an extension with a weight of ten pounds was applied to the limb. Skiagrams were taken (Figs. 324 and 325).

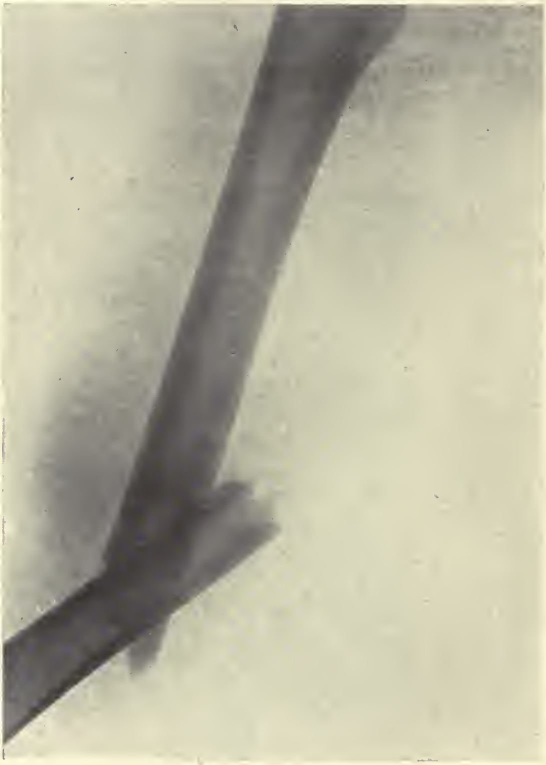

Fig. 325.—Same as preceding figure (lateral view).

COMMENTS AND OPERATION

Dr. Murphy (November 3, 1914): [Incision and exposure of fragments.] There is the hematoma about the site of fracture. Note how the bone is fragmented, and how hard it is.

He has a great muscular, as well as bony, development for a boy of his age. [The fracture was reduced and plated (Figs. 326 and 327) with the same technic as in the previous case.]

RECAPITULATION

Let the record show that the field is perfectly dry after inserting the plate; that the entire operation was performed without hand-contacting; that there was great difficulty in accomplishing the reduction; that it required the entire pressure of the lever to straighten the fragments; that the fracture was reduced without complications and good alinement was obtained; and

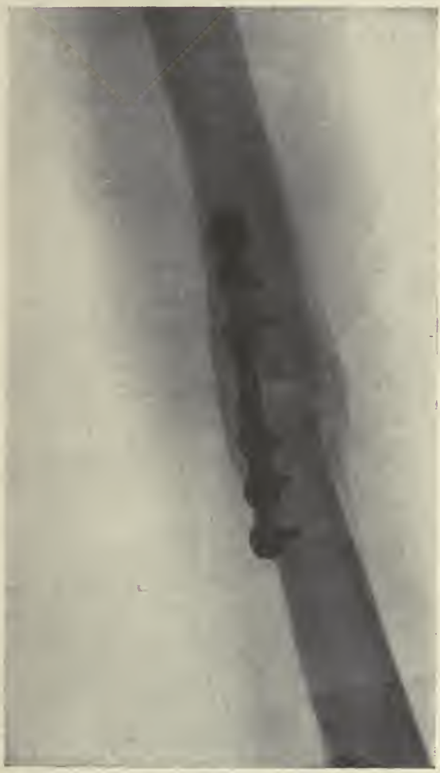

Fig. 326.—Postoperative skiagram of fracture of shaft of femur showing Lane plate *in situ*. Note extensive subperiosteal callus formation (anteroposterior view, taken six weeks after operation).

that a six-screw plate was inserted and a plaster-of-Paris dressing applied.

There will be great tension upon the plate.

POSTOPERATIVE COMMENTS

We operated upon this patient two weeks after the fracture was received, after having made a number of futile attempts at reduction and retention of fragments—one under anesthesia. The luxation recurred each time. By the end of two weeks the tissues about the fracture have attained their maximum powers, both of resistance against bacterial invasion, and of regenerative

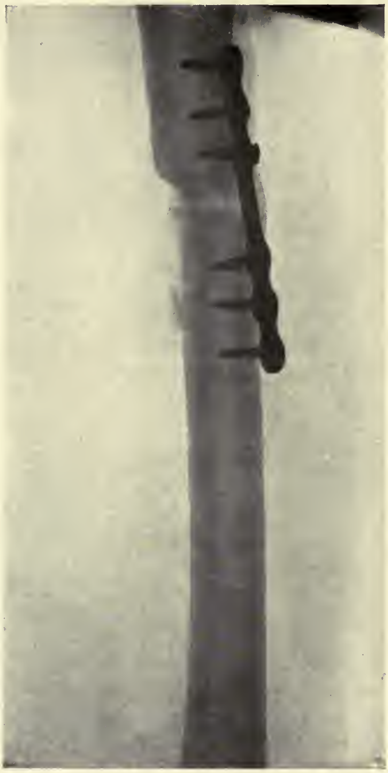

Fig. 327.—Same as previous figures (lateral view). Note absence of deformity and note also good alinement.

activity. Immediately after the accident, on the other hand, the opposite conditions obtain. You all know how much more difficult it is to infect a granulation-wound than a fresh wound.

[Cases of fracture of the lower third of the femur have been previously published in the Clinics December, 1912, pp. 853–860; August, 1913, pp. 617–624, in which phosphor-bronze plates were used and Dr. Murphy's bone-skid is illustrated; June, 1914, pp. 545–556, in which the fracture was compound and the lower end of the upper fragment penetrated the knee-joint and rested under the patella.]

[*Note.*—There was complete primary union of soft parts and bone. The end result is reported as excellent.]

RIGHT KNEE: LUXATION WITH FRAYING OF INTERNAL SEMILUNAR CARTILAGE AND OSTEOPHYTE—ARTHROTOMY—EXCISION OF SEMILUNAR CARTILAGE AND OF OSTEOPHYTE. *LEFT KNEE:* HYPERTROPHIC OSTEOARTHRITIS. TALK ON ORIGIN, NATURE, AND TREATMENT OF BONE AND JOINT INFECTIONS

Summary: Clinical history of patient; comments—primary foci of acute metastatic arthritides; interpretation of the skiagrams; analysis of history; classification of the metastatic arthritides; luxation of patella confused with luxation of semilunar cartilage; operation—internal semilunar cartilage loosened in the middle and frayed out at the edges; no-hand-contacting of joint; great strength and tensile power of fibrous capsule; the fibrous capsule as the "leather-bottle" of the joint; recapitulation; postoperative comments—coffer-damming of joint preparatory to operation indicated, especially in the aged. Talk on Dr. Murphy's plan for treating joint-infections; significance of chill in prognosis of joint-infection; bacteriology of joint-infections; "defensive pus"; earlier contradictory bacteriologic findings in Neisserian infections; experiments with pneumococci; value of Buck's extension; two sources of pressure in an inflamed joint; treatment of acute metastatic osteomyelitis; the wrong treatment of an acutely infected joint *vs.* the right method; effects of treatment; preparation of vaccines; the ancient Venice turpentine "quasi-vaccine" method.

CLINICAL HISTORY OF PATIENT

The patient, a female aged sixty-three years, was admitted to hospital June 8, 1914. The family and past histories were negative.

When the patient was eight years of age she fell and struck the right knee upon a broken bottle, cutting a gash just internal to the patella. From that time until sixteen years ago (1898) the right knee often became locked so that she could not walk, compelling her to go to bed for relief. The knee sometimes remained locked for from three to ten days at a time. When the "unlocking" occurred, it took place during sleep, and it was for this reason that she went to bed whenever the locking happened. During these attacks the knee was painful and always tender. The locking was especially prone to occur whenever the patient threw the leg quickly or violently to the right, or in rising quickly from a sitting position.

Twenty years ago (1894) she was in a runaway accident and was thrown from a carriage, falling with her full weight upon both knees. Soon after this incident a grating sensation developed in the right knee on both active and passive motion. This grating has gradually increased in intensity and frequency until now it is constant, and accompanied by considerable pain. The knee often puffs up, but never becomes red. The patient has never had chills or fever with the swelling of the joint. The pain in the knee becomes particularly severe if she rotates the body without moving the feet.

Six years ago, in 1908, the *left* knee began to bother her, but in a different manner—by grating but *never locking*, and causing pain on movement. There is a "drawing feeling" on the back of the knee and in the muscles of the thigh, and the knee has gradually become weaker with the progress of the affection. At present the patient cannot bend the knee sufficiently to climb stairs.

About one year ago she consulted a physician who told her that she had a growth in both knees and that the knee-cap was roughened. This roughening, he said, was the cause of the pain. He also informed her that she had "kidney trouble."

COMMENTS

Dr. Murphy (June 11, 1914): The left knee shows a typical hypertrophic arthritis. The sequence in the right knee was trauma first, then luxation of the internal semilunar cartilage, then the development of traumatic osteoarthritis with the periosteal and cartilaginous ossifications which one sees here in the skiagrams (Figs. 328 and 329), and which have given her so much trouble. Undoubtedly the underlying trouble in both knees is of the same type—metastatic arthritis from some focal infection. These metastatic infections are very frequently associated with chronic alveolar abscesses when the arthritis is of the slowly progressive, chronic type. The acute type of metastatic arthritis, on the other hand, has its origin, as a rule, in a nasopharyngeal and occasionally a tonsillar infection. I have sent for the skiagrams of a recent case which is almost the counterpart of this one. The frontal skiagram of that patient's knee is almost identical with the frontal picture of this knee.

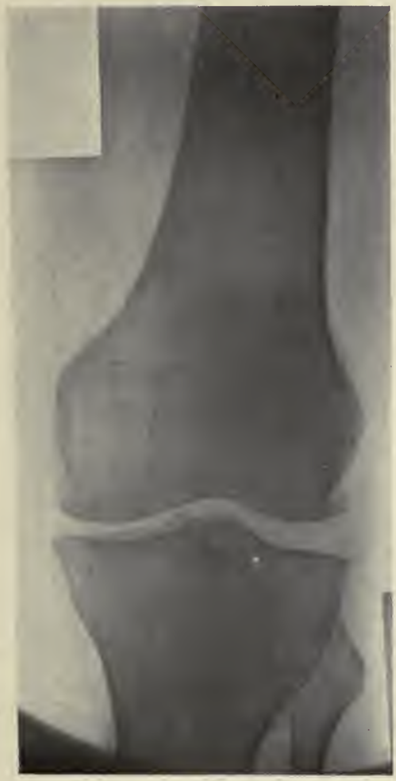

Fig. 328.—Preoperative skiagram of right knee-joint, showing traumatic osteoarthritis. Note "lipping" of articular edges of femur and tibia (anteroposterior view).

At present the condition of the semilunar cartilage is not so important to the patient as the secondary complications which have developed in the knee. See the osteophyte which projects from the upper surface of this patient's tibia into the knee-joint! (Fig. 329).

In the other patient's knee the intra-articular osteophyte had its origin from the external tubercle of the spine of the tibia and projected into the center of the joint: in the patient before us this bony spicule is located on the anterior lip of the tibial surface (Fig. 329). The other patient also gave a definite history of trauma to the knee, which appeared to stand in causative relation to the subsequent changes. Twenty-five years ago he received a stab-wound in the knee, almost or actually at the site

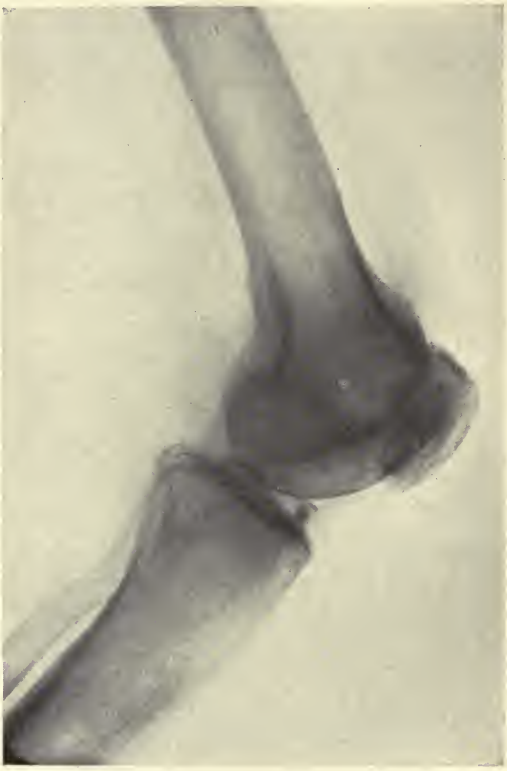

Fig. 329.—Same as preceding figure (lateral view). Note large osteophyte springing from anterior margin of articular surface of tibia.

where the osteophyte developed later. The osteophyte has, in fact, the exact appearance of a dagger-tip, so that, had the skiagram not shown the projection to be bony in structure and in density, we might have thought that a piece of the knife had been left in the joint.

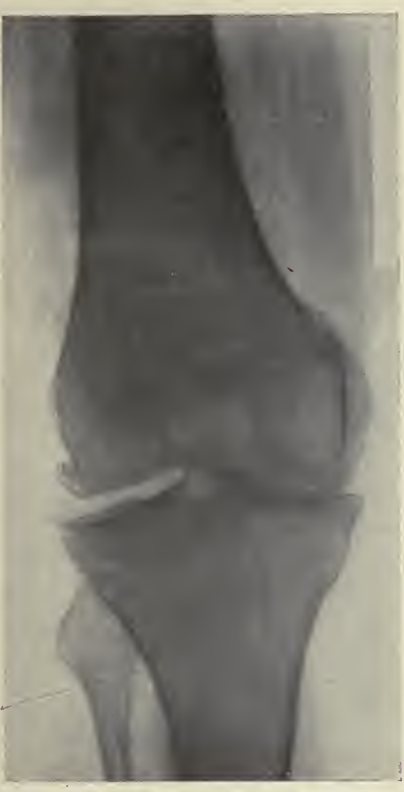

Fig. 330.—Hypertrophic osteoarthritis of left knee-joint. Note "lipping" of external condyle of femur and of both tibial tuberosities. Note also obliteration of space between internal condyle of femur and internal tuberosity of tibia, owing to partial destruction of internal semilunar cartilage (anteroposterior view).

Let us analyze the history of our patient. First she received the bottle-trauma to the right knee-joint, and this trauma caused laceration and luxation of the semilunar cartilage. As the result of the irritation of the displaced cartilage there developed traumatic synovitis, which, by its recurrence, produced thickening and then chondrification of the capsule. The capsule became more and more cartilaginous and thickened, until finally an ossification developed in the base of the cartilage. It is not until such extensive pathologic changes have developed that most of these cases of injury to the semilunar cartilage fall into our hands.

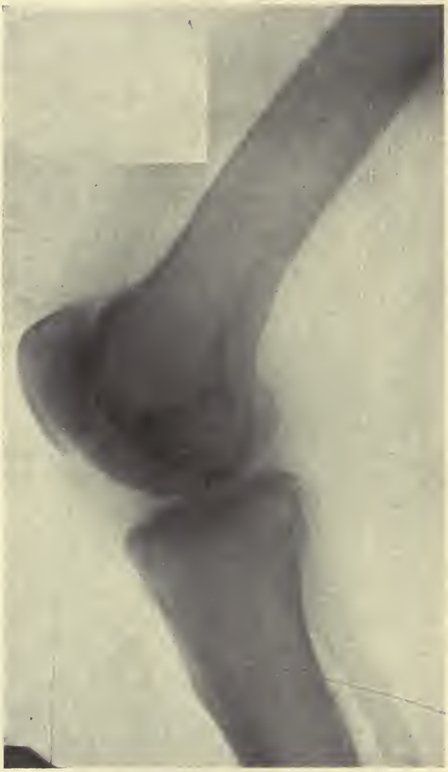

Fig. 331.—Hypertrophic osteoarthritis of left knee-joint. Note internal condyle of femur resting directly upon internal tuberosity of tibia (lateral view).

Here are the skiagrams of the left knee-joint (Figs. 330, 331). You see an absorption of all the cartilage over the internal tuberosity of the tibia, and that the internal condyle of the femur has descended until it is lower than the one on the right side (Fig.

330). In the *left* knee-joint *hypertrophic* arthritis has developed —a condition which differs entirely from that on the side upon which I am going to operate. Upon the upper surface of this left knee-joint the cartilage has disappeared entirely.

The lesions that in the early days were classified as rheumatoid conditions we now believe are septic conditions. In the work on the joints that has lasted over a period of twelve years, or since we performed our first arthroplasty, and thirteen years since we began the preparation of the work, we have begun to recognize the sources of infection that produce definite and clean-cut types of secondary lesions.

We divide arthritides, first of all, into the acute type. The source of infection in the acute type is first and most commonly the pharynx; secondly, the nasal mucous membrane; thirdly, the urethra (gonorrhea); fourthly, the tonsils. While the tonsil has been most feared as the source of acute metastatic arthritis, the pharynx is in reality the commonest source. When the infection is confined to the tonsil alone, there rarely arises an acute metastatic arthritis.

Furthermore, we have learned that there are definite days upon which these metastases fall due. Once, in checking up our Neisserian infections, we found that 95 per cent. of the metastatic arthritides in gonorrhea occurred between the eighteenth and the twenty-second day. One patient attributed his sore joint to exposure to inclement weather; another, to his having caught cold; the third, to his having jumped over a fence, and the fourth to something else: the clinical fact remains, however, that it was the coincidence of the event with the completion of the evolution of the metastatic gonorrheal arthritis that misled the patient. From this you see that the evolution of gonorrheal arthritis has a definite course, and that it is not an accident to which these metastases have heretofore been charged. So, too, in typhoid fever a typhoid joint-metastasis rarely occurs before the end of the third week: most of them occur between the fourth and sixth weeks. Pneumococcic infections and those of the grip type occur practically always between the eleventh and fourteenth days after the onset of the infection: initiated by a chill, one or more joints may be involved in the metastases at this definite time.

The second division of arthritides is the slow, gradually destructive type, and we speak of these as acute or chronic rheumatoid arthritides, with special reference to the chronic, which is called hypertrophic (rheumatoid) arthritis. The large majority of the cases of hypertrophic arthritis is associated with the infections of the alveolar type or abscesses at the roots of the teeth. I have a man now coming to the office whose attacks of arthritis coincided with his attacks of tooth trouble. He had six teeth drawn and the roots of all were so diseased and had such offensive pus that you could hardly stay in the dentist's office while the teeth were being extracted. Of course, the abscesses were present when the teeth were defectively filled, otherwise they would not be there now.

We are now, in our cases, checking up to find out just what type of infection started a given case of arthritis. We are getting results in proportion to the accuracy with which we arrive at the source of the infection when using vaccines. There is no vaccine that will benefit this patient's knee-joints: it is like calling the fire engine to restore the furniture after a fire has destroyed it.

There is another lesion with which luxation of the semilunar cartilage is very frequently confounded, and that is luxation of the patella: this mistake had been made in practically every case of luxation of the patella that has come to us. Thus, in nine cases of luxation of the patella all were diagnosed as recurrent luxations of the semilunar cartilage, with the exception of two, of which one was congenital and the other a case of double luxation, which was pathologic. The reason for this mistake is that in their symptoms luxations of the patella mimic recurrent luxations of the semilunar cartilage, and the physician rarely ever arrives before the luxated patella is replaced.

OPERATION

[Incision.] I want you to note the point we have made in connection with the lodgment of metastatic infections in the joint tissues, namely, that the infection does not select the free cavity of the joint, nor the surface of the synovial membrane, but lodges in this tissue I am exposing now—the subsynovial layer of the joint-capsule—in which are located the vessels and the lymph-spaces. The subsynovial layer is sandwiched between the synovial membrane and the fibrous capsule proper.

[Cuts through the synovial membrane.] Now I am down upon the tuberosity of the tibia. Here is the internal semilunar cartilage, and you see that it is loosened in its middle: see the hypertrophied villous mass that insinuates itself between the bone-ends and which by its irritation keeps up a recurrent inflammation! Now I want to show you the frayed edges of the cartilage as I remove it: that part of the semilunar cartilage has "gone to a frazzle"; it has been subjected to pressure all the time. [Removes internal semilunar cartilage and demonstrates it.] There is the part that was inside of the joint; there is that hypertrophied villous mass that extended clear across the midline of the joint; there is the osteophyte which you saw in the skiagram (Fig. 329)—it is merely the sequence of the constant traumatic irritation. You can see how the chronic synovitis has affected that bony surface which has been absorbed beneath the cartilage.

You will note that we never let anything into that joint that had contacted with our hands, or contacted with anything that had touched the hands: this is the key to the prevention of infection, and therefore to the ultimate results. The most difficult part of this technic is to tie the catgut with instruments. A man in Australia sent me two instruments for this purpose, but they were just as awkward in my hands as chop-suey sticks, yet he could use them most dextrously.

I am now bringing back and reuniting the edges of the synovial membrane: we have endeavored not to traumatize its under surface at all. Next, I close up the fibrous capsule of the joint. . . . This fibrous capsule does not look like a tough, fibrous membrane, but in our experiments we found that it withstood an hydraulic pressure of from twenty to one hundred and forty pounds before it ruptured. Its great strength is due to its absence of vascularity; its great tensile power accounts for retention of the products of infection within the joint for such a long period of time, differing from the comparatively shorter retention of the products of infection in the cellular or soft structures, the strength, tension, and resistance to infective destruction of which are much less.

In the soft parts you get an infection that slowly destroys the tissues and soon comes to the surface. On account of its non-vascular condition the fibrous capsule remains like a leather bottle, retaining the products of infection within the joint under a certain amount of tension for weeks and months at a time. When you analyze the destructive effects of infection in a joint you must take into consideration the factors that obtain in other tissues. First, you must consider that the destruction is produced by the toxic and the biotic effects of the microörganisms, the extent depending upon their virulence; secondly, that the destructive effect of infection is enormously increased, all other things being equal, by the tension under which the products of infection are held.

There is no bleeding from the operative field.

RECAPITULATION

Let the record show that the outer margin of the internal semilunar cartilage was frayed and spread out over the joint; that there was a hypertrophic villous mass beginning in the mid-line and extending into the joint; that in removing this mass the dissection extended over to the left side, as well as to the right side, of the ligamentum patellæ; that the internal semilunar cartilage was excised; that the tubercle of the ossifying cartilaginous new-growth was chiseled off; and finally that the limb was dressed in extension with a weight of 20 pounds applied to the Buck's apparatus. Let the record show further that the operation was performed without hand-contacting.

POSTOPERATIVE COMMENTS

At her age patients have little or no resistance against infection. Senile arteriosclerotic changes are common, and herald the diminished resistance to every type of infection. Ten days ago our patient had an intra-articular injection of 2 per cent. solution of formalin in glycerin to produce a chemical inflammation to coffer-dam the lymph-spaces and to stimulate polymorphleukocytosis, thus making the interior of the knee-joint resemble a granulating surface more than a fresh, glistening synovial membrane.

The Buck extension apparatus will be retained two weeks, and at the end of this time we shall gradually lighten the weight until we remove it entirely.

Here is another patient who had almost an identical condition in so far as the recurrent synovitis is concerned, but he did not have a displaced semilunar cartilage: he had a stab wound of the knee-joint. He is about ready to go home, and we are just beginning to let him walk. It is four weeks since he was operated upon. He did not have metastatic arthritis, but chronic mechanical synovitis due to constant irritation of the surface of the joint by the osteophyte shown in the picture. He got up about three weeks after the operation. You see he walks around in the daytime: we leave the Buck extension on at night.

TALK ON ORIGIN, NATURE, AND TREATMENT OF BONE AND JOINT INFECTIONS

In the management of these joint-infections we have outlined a fairly definite plan. In the first instance, we regard every case of acute arthritis as a surgical lesion that is secondary to a primary focus of infection in some other part of the body. In other words, we consider that acute arthritides are lesions of infection, rather than of metabolism. That the metabolic changes have an influence in the course of some of these joint-diseases is beyond question; but in the case of acute arthritis, the lesion is due to an infection. When metastatic involvement of a joint is initiated by a chill, we always estimate that one joint at least will have bony ankylosis as a sequence of the infection, unless it be managed along other lines than those along which such joint infections have been managed in the past.

I remember, in one of my very early lectures, when I was an intern in the Cook County Hospital, that Dr. Hollister made this differentiation between the types of arthritis: at that time pyemia was very common and developed from numerous infections that were improperly treated. He said to me: "Doctor, that patient hasn't pyemia because the arthritis did not begin with a chill; he has rheumatism." Though a medical practitioner, Dr. Hollister had learned, even at that time,—one-third of a century ago,—to differentiate the severe arthritic cases that came directly from the septic wounds which prevailed in those days, and which, forecasting from the initial chill, would eventuate in great disability to the patient. We have just reverted to that same principle, which holds with equal force at the present time. The patient who develops an infection, then a chill, and then an arthritis or multiple arthritides, is going to have ankylosis of one or more of the large joints.

Except in the streptococcic type, the bacterial emboli lodge in the subsynovial vascular and lymphatic tissue which lines the fibrous layer of the capsule. Because the bacteria lodge on the "wrong" (external) side of the synovial membrane no microorganisms are found in early aspirated fluid, and their absence from the joint synovial fluid but corresponds with our experience with the abdominal synovial fluid. For a long time, in every case of acute appendicitis when infections exist close to the peritoneum, but have not ruptured into it, we were swabbing out the pus which, upon bacteriologic examination, was reported as sterile. The microörganisms had not left the local focus of infection, nor penetrated the peritoneal serous membrane, and that creamy serum that proved sterile was "defensive pus"—lymph, full of lymphocytes and full of polymorphs ready to pounce upon attacking bacteria. Exactly the same sterility of the effusion occurs in the joints. In the early stages the aspirations are negative time and again until finally one day you aspirate the fluid and find microörganisms free in the joint: that, however, is usually in the later stages of the disease.

You will recall the contradictory results obtained in the earlier aspirations of Neisserian joint-infections. One investigator aspirated the synovial fluid early and found no gonococci;

another aspirated later and found gonococci; a third aspirated still later and found none, as all had been removed from the fluid of the joint through the phagocytes by this time, but they still existed in the tissue. None of these investigators had learned that the finding of the microörganisms depended upon the proximity of the time of aspiration to the time the metastasis took place. These findings were at first very misleading, because the infection was then supposed to be a surface infection of the synovial membrane, whereas now we know that the bacterial emboli lodge beneath (outside of) the synovial membrane that immediately covers and protects the joint.

We have had some very instructive experiments made by Dr. Schrager in connection with the pneumococcic infections, which are very virulent in animal joints, particularly in dogs. He injected a half-syringeful of pneumococci into the front knee-joint of a dog without traumatizing the joint. Next, using the same fine hypodermic needle, he injected the other knee-joint with the remaining half-syringeful, at the same time extensively scratching and irritating the synovial surface with the needle. In this traumatized knee-joint he produced a phlegmon that was fatal for the dog. The joint that was not traumatized underwent complete resolution without incapacitating the dog at all. These experiments show the rôle that trauma to the synovial surface of the joint plays. So, also, if you disturb the vascular subserous tissue that lines the synovial membrane of the joint the same intense infection arises. These experiments were performed in the very early days of our joint-investigations, and helped establish a very great respect for every type of joint-trauma. It took us twenty-five years to learn similar facts concerning trauma of the peritoneum: every time one traumatizes the peritoneum one increases the danger of bacterial peritonitis.

In the management of these cases of acute arthritis we apply traction by means of Buck's extension to relieve the intra-articular pressure due to the spasmodic contraction of the muscles. Infection destroys not alone by its bacterial toxins, but also by this increased intra-articular pressure. In the joint there are two different sources of pressure—that from the tension of the joint-exudate, and that from the immobilizing or guarding contraction of the regional joint-muscles. In infections elsewhere than in joints there is only one source of pressure.

In the treatment of metastatic osteomyelitis, what should one do within the first twenty-four hours? Trephine or vent the bone to relieve the tension and, if opened within the first twenty-four hours, no necrosis will result, so that curetting is unnecessary, and the bone heals right up. What is the wrong way of treating an infected knee-joint? By applying a poultice and waiting six weeks with the products of the infection in the joint held under double pressure—the capsular pressure and the intra-articular pressure—until the cartilage of the joint is destroyed.

The proper method of treating an acutely infected joint consists of two measures: first, apply an extension-apparatus and apply it *immediately;* that relieves the pain, lessens the liability of destruction of the intra-articular cartilages and of the synovial membrane, and prevents deformities and luxations; secondly, aspirate to relieve the tension of the infection-products.

We have in the hospital a most beautiful illustration of a metastatic infection in the wrist-joint of a patient whose temperature was 103° F. We aspirated once only, and the synovitis disappeared rapidly. There was "defensive pus"—when we came to look for microörganisms none was found. This "defensive pus" indicated that the subsynovial vascular layer of the capsule was infected, and then, in order to supply an additional means of defense, we injected the joint with a 2 per cent. solution of formalin in glycerin. Formalin produces a polymorph leukocytosis when injected into a joint.

Effects of Treatment.—Aspirate and if necessary re-aspirate the joint to keep down the tension in the capsule, thereby lessening the amount of destruction of the non-vascular synovial layer, and then inject it with a 2 per cent. solution of formalin in glycerin to make the fluid in the joint sterile, or a poor culture-medium, and to increase the polymorph leukocytosis, which factors mean increased resistance.

What is the end-result of this mode of treatment? In the first instance, let us state that the joint may and often does advance to the stage of bony ankylosis. Provided that the limb has been kept perfectly straight during the use of Buck's apparatus, bony ankylosis in the extended position results in a limb that is of the greatest service to the patient, in that the pain has been relieved and a good conformation of the joint is maintained. I can show you joints that have assumed all sorts of fantastic shapes. In the acute infections the knee-joint assumes the position of flexion by preference because in that position the joint accommodates the greatest quantity of fluid, and if allowed to go on, the leg flexes upon the thigh and becomes ankylosed in flexion. Such deformity may be avoided by the early application of extension.

Why do I lay stress upon the importance of preventing deformity? Formerly, the courts held us responsible for the deformities caused by fractures: I think that in the future we are going to be held absolutely responsible for the deformities caused by the arthritides as well, because such distortions are unnecessary and always easily avoidable.

In all these acute cases we endeavor to isolate tne microorganism that produces the infection, and we then prepare and administer vaccines from the very beginning. If we cannot produce an autogenous vaccine we estimate the source and path of the infection, and then take a mixed vaccine of the type that produces a like infection. If the source be the pharynx—from infection of which metastatic arthritis is most commonly produced—then we make cultures at the time. If you give freshly prepared mixed vaccine and get a pronounced reaction in the way of fever and leukocytosis, that patient is going to be benefited by the injections, because you have approached the type of his infection. The same benefit may be predicted with tuberculin. If you give a dose of tuberculin to a patient who has no tuberculosis, the drug would have no more effect upon him than if it were so much water: he might have any other type of infection, but tuberculin has not the slightest constitutional effect upon any non-tuberculous infection. As we interpret it from a clinical standpoint, the greater the reaction the closer we are to the variety of microörganism which caused the infection. The theory might be entirely wrong, but it at least affords a working basis, and our clinical observation shows that those patients in whom the reaction is pronounced derive the greatest benefit. This clinical observation is not new, either.

Thirty years ago there lived in Liverpool a physician who advocated producing aseptic suppurations in distant portions of the body for the relief of arthritis. He injected ordinary Venice turpentine into the loin or the buttock to produce a sterile abscess, and then at the end of four or five days he opened the suppurating focus and the patient got well. That physician did not know the first rudiments of bacteriology, nor of vaccines, but from clinical observation he found out that he got results. The abscess did not consist of the débris caused by bacterial infection, but merely of serum teeming with polymorphonuclear leukocytes—practically 100 per cent. polymorphs. It also produces a constitutional leukocytosis which probably was the most potent factor in the relief of the arthritides. This constitutional leukocytosis is similar to that caused by the formalin and glycerin injections. Undoubtedly his clinical observation was accurate; it is quite in consonance with our more recent knowledge of vaccines and their effects.

[Cases of fracture or luxation of the semilunar cartilages have been previously published in the Clinics as follows: December, 1912, pp. 861–866 (fracture); February, 1913, pp. 47–51 (luxation); *ibid.*, pp. 53–57 (floating, with illustration of cartilage); June, 1913, pp. 435–441 (luxated and displaced across median line of joint); February, 1914, pp. 151–160 (fracture of cartilage, associated with luxation of patella—illustrations of operation for latter condition); December, 1914, pp. 1151–1157 (fracture with exostosis—the "penknife" case referred to above); August, 1915, pp. 779–787 (fracture, with villous synovitis—illustrations of cartilage). (See also the next case of the present number.)]

[*Note.*—There was complete primary union and the patient made a good recovery.]

FRACTURE OF INTERNAL SEMILUNAR CARTILAGE — ARTHROTOMY — EXCISION OF CARTILAGE

Summary: Clinical history of patient; comments—varieties of internal luxation in knee-joint; pathology of internal luxation; development of rice-bodies in tuberculosis of joints; method of completely exposing knee-joint; palliative treatment; immediate operative treatment; operation—excess of synovial fluid; semilunar cartilage fractured, frayed, and flattened, and incorporated with hypertrophied villi; cartilage excised; method of suturing capsule and then skin; no-hand-contacting operation; postoperative comments—infection of joint *vs.* peritoneal serosa; perforative peritonitis *vs.* subperitoneal infection; resistance of skin *vs.* synovial membrane to abrasion and infection; infection of subsynovial layer of capsule; "defensive" pus; phagocytosis of Metchnikoff; use of Buck's extension to combat postoperative traumatic reaction; use of 2 per cent. formalin in glycerin; coffer-damming of tissues a barrier to infection; additional note four months after operation.

CLINICAL HISTORY OF PATIENT

The patient, a male aged fifty years, was admitted to hospital September 9, 1914. The family history reveals that one sister died of pulmonary tuberculosis. The past history shows that he had pneumonia at thirty years of age and again at thirty-nine.

The accident that led to the present trouble was received thirty-five years ago. While kicking at a football the patient missed the ball and fell. He immediately felt a severe pain in the right knee-joint on the inner side, which was so acute that he felt faint. He could not flex the knee, nor could he walk. Fifteen minutes later, on attempting to get up, he felt much relieved and was able to walk. Four hours later the knee was slightly swollen. The soreness lasted about three days.

Since that time the patient has had at least twelve attacks, occurring at different intervals and upon different occasions. He had one attack while getting out of bed in the morning: upon putting the right foot down, he noticed a severe pain as the knee moved, but by turning the leg in different directions the pain was relieved. The last attack occurred three weeks ago, when the knee gave way with pain. Since that time he has not been comfortable when walking or when standing (Figs. 332 and 333).

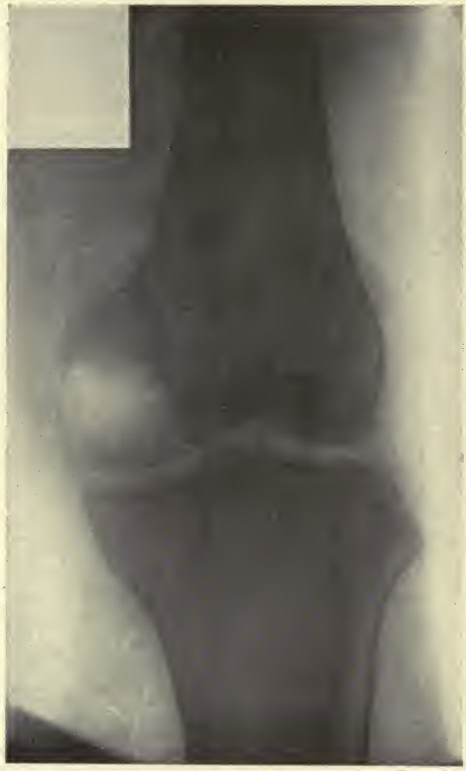

Fig. 332.—Preoperative skiagram of case of luxation of right internal semilunar cartilage. Note absence of osteophytic joint-changes. Compare to previous case (anteroposterior view).

COMMENTS

Dr. Murphy (September 11, 1914): Where did the patient have most of the pain?

Intern: On the inner side of the knee-joint.

Dr. Murphy: On the inner side, which is the most common position for the pain with internal luxations of the knee-joint.

Of the luxations that occur within the knee-joint, you have first and most commonly the subluxation or fracture of the internal semilunar cartilage; secondly, subluxation or fracture of the external semilunar cartilage: the internal is luxated or fractured fifty times to the external's once; thirdly, the laceration or fracture or avulsion of the tubercles of the tibial spine, to which the crucial ligaments are attached, and following the avulsion the development of exostoses at the sites from which the bone has been pulled off by the crucial ligaments: these exostoses impinge against the femur and cause the patient considerable trouble; fourthly, the loose interarticular floating cartilage, of which there

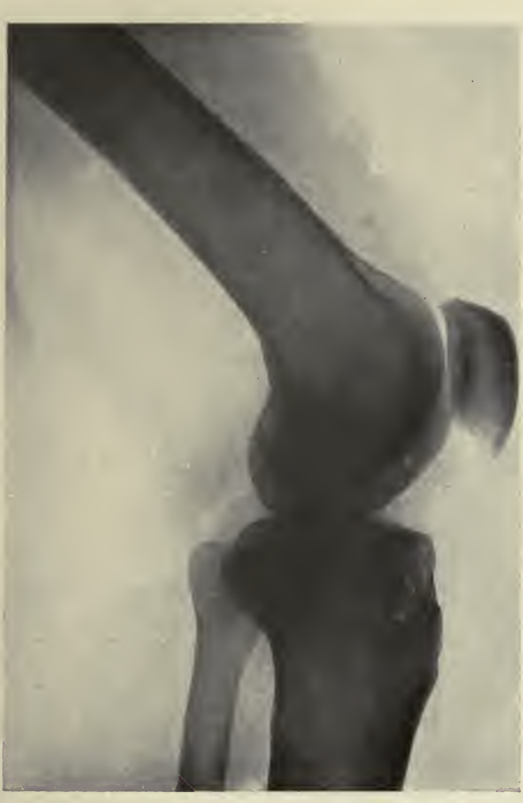

Fig. 333.—Preoperative skiagram of case of luxation of right internal semilunar cartilage (lateral view).

are two varieties—in one the cartilage is partially detached and movable, while in the other the cartilage is completely detached and floating: the former variety is the easier to control and the easier to remove, because the partially detached cartilage remains within a definite range during the operative approach to it.

The manner of pathologic growth of these cartilages is very interesting. They appear to grow practically the same as osteophytes grow: at first small, with the continued irritation, proliferation appears upon the surface: whether this proliferation begins on the surface or whether it is an extension from within has not been definitely determined, but it is believed to be formed from the stretching and straining due to recurrent trauma.

All these varieties of luxations within the knee-joint produce serious pathologic changes within the joint. By the recurrent trauma they produce first, congestion; secondly, fixation of the joint; thirdly, swelling and edema of the villi of the synovial membrane. As these villi become detached they finally get caught between the joint-surfaces, become traumatized, and afford additional sources of trouble. Then, if you keep up the trauma, there is a tendency to the precipitation by the trauma of infective microörganisms, most commonly embolic from a primary focus of infection in some other portion of the body, and less commonly tuberculous.

When tuberculosis is deposited in the joint synovial membrane of adults, you have the greatest amount of trouble. In these cases there is as a sequence the development of a large swelling. The villi become elongated to the size of a grain of rice, a grain of oats, or a grain of wheat, and some even larger. They are later detached and then are known as rice (oryzoid) bodies in the joint. The late Dr. Fenger said that all rice-bodies were of tuberculous origin: we do not believe this to be the case. We believe that rice-bodies begin in hyperplasia of the villi, and that the detachment of the hyperplastic villi forms the rice-bodies. That rice-bodies are *sometimes* associated with tuberculosis is beyond question.

The continuance of recurrent trauma eventuates in complete impairment of the joint. You find greatly impaired joints of this variety more commonly in stout adults and in women. The pinching of the villi between the joint-surfaces leads to the production of synovitis. The fibrous portion of the capsule distends, while the subsynovial portion of the capsule becomes thickened, even to the extent of from one-half to three-fourths of an inch. There is an area of edema outside of the fibrous capsule.

The question arises as to the best method of dealing with such traumatized joints in fat people—whether to open the joint entirely and clean out the whole synovial membrane, exposing it by splitting the quadriceps tendon, the patella, and the ligamentum patellæ (CLINICS, October, 1915, Figs. 253–259, pp. 1000–1003), and then closing up by bringing the edges of the quadriceps tendon, the patella, and the ligamentum patellæ together. This (modified Corner's) method affords opportunity for a full exploration of the joint with the least manipulation—a very important desideratum. You will recall that in my explorations of the knee-joint performed by splitting these three structures I stated that the only structure which must be split with care is the patella: it is split in its long axis into halves, and each half is retracted to its own side. This method gives an ideal exploratory exposure.

In the palliative treatment of these cases, as it is performed to-day in general practice, the plan is, in the first instance, to put the part at rest. Later, when the patient is able to be up and about, a leather knee-case is applied and retained for months or even years. This conservative method occasionally relieves the condition permanently, but if the semilunar cartilage is fractured or frayed—an injury of the knee-joint which Rutherford Morison finds to be by far the commonest—then the method does not relieve the condition at all. In the laboring classes in England, particularly at Newcastle, where the miners work with the shovel in a stooping position, it is a common thing for a miner to throw out the internal semilunar cartilage. When this accident occurs, the patient is sent to hospital, and as soon as the reaction from the trauma has established itself, the joint is opened and the semilunar cartilage excised, and the patient is back at work within three weeks. When I was at Newcastle three years ago Morison showed me over 500 specimens of excised semilunar cartilages—a number that conveys to you some idea of the frequency of this injury.

OPERATION

[Incision.] There is the fibrous capsule of the joint: see it shining as a membrane! Inside of that fibrous capsule is situated the tissue to which we have called attention so many times—the subsynovial vascular tissue, where the bacterial emboli in cases of synovitis find lodgment. You see there is plenty of it in this case. Owing to the presence of connective tissue from the recurrent trauma it is difficult for us to separate this subsynovial layer. See how edematous it is! It is infection involving this loose periarticular tissue that raises such havoc when virulent in type. Now the synovial fluid, of which there is an excess, is escaping. There you see the internal semilunar cartilage, and that it is movable. The constrictor has not been applied properly in this case so that we shall remove it entirely. Wherever you do not obtain complete arterial stasis, but produce venous stasis, then the tissues bleed more profusely with the constrictor on than with the constrictor off. [Removes constrictor.] Now you see that the venous hemorrhage has stopped, and that there is a slow return of arterial bleeding.

That cartilage does not look like a semilunar cartilage at all. From being white and glistening it has become a vascular body. There is the line of fracture of this cartilage. We shall excise it together with the fatty tissue around it. Let me show you another change. In place of the fractured end of the cartilage being smooth it is ground between the bone-ends and pinched until, as usual, it has become flattened and its edge frayed out.

Let the record show that in much of its extent this cartilage does not resemble a semilunar; that it is fractured circumferentially and that from the repeated grinding hypertrophied villi have become incorporated with it.

We shall gently brush out the blood from this field so as to obtain a clear view of this deep joint-space. You see what a perfect exposure I have made. I shall remove all these fragments now.

Let the record show that the internal semilunar cartilage was found to be completely detached from the tibial tuberosity, and that the fractured end projected between the bone-ends for one-third of an inch.

See where the cartilage was free—down on one side! The absence of attachment below shows how it was luxated.

Let the record show that the internal semilunar cartilage had so changed in appearance as to resemble vascular tissue.

[Sutures incised edges of synovial membrane.] That suture closes the synovial portion of the joint-capsule and, by everting the edges, prevents their turning in and becoming a source of trouble subsequently. Now I close the fibrous portion of the capsule. In beginning this cutaneous suture you notice that we insert the first stitch in the middle of the curved incision: had we started at the end, the apposition would not have worked out evenly, but by starting in the middle and then working toward each end, there is obtained a good cosmetic effect.

Let the record show that after approximating the edges of the synovial membrane and of the fibrous capsule accurately, the skin was closed with horsehair sutures; that no ligatures were used; and that there had been no contacting of the field of operation either with the gloved hand or with instruments at the sites where they had touched the gloved hand.

POSTOPERATIVE COMMENTS

The strict observance of asepsis is the key to all joint-work. Remember that in operating upon joints you have an entirely different proposition from that of operating in the peritoneal cavity, which we have always regarded heretofore as the most dangerous place for infection to lodge in. But in connection with the peritoneum there are two types of infection, and one is the surface-infection of the peritoneum which comes from perforation into the free peritoneal cavity, while the other is the subperitoneal type. The *perforative type* of infection is never so dangerous as the *subperitoneal.*

What do we mean by the subperitoneal type of infection? It is the type of infection that occurs after parturition and after abortion. When you say that you have postparturient peritonitis or postabortive peritonitis, that is not what you mean: you mean that you have postparturient or postabortive *subperitoneal* infection. The infection is not *upon* the surface of the peritoneum at all. What is the portal of entry in subperitoneal infections? The infection enters through the unhealed lacerated cervix; through the raw surface of the uterus at the site of the placenta after its detachment, or perhaps through an abraded surface of the uterine cavity. Where do the lymphatics from both these portals of infection lead to? They do not empty into the peritoneal cavity at all, but into the subperitoneal tissue between the layers of the broad ligament, the infection spreading *under* the peritoneum, and *not* invading the peritoneum itself. If you open the peritoneum in these cases—and Dr. Bridge, who is present to-day, will recall the autopsies upon which these statements are based—he will remember that we used the term dry peritonitis—dry because the infection was not upon the surface, but *beneath* the surface. It does not benefit the patient to operate upon and drain, in a case of post-parturient peritonitis, because you do not succeed in reaching the area involved in the infection.

Infection in the subperitoneal cellular tissue is thus entirely different from the *intraperitoneal* infection that is due to perforation of an appendix or of an ulcer of the digestive tube. In perforative peritonitis the infection is *upon* the surface of the peritoneum, and the peritoneum offers great resistance to the spread of the infection, just the same as the synovial membrane resists infection if not previously subjected to trauma.

In this case Buck's extension apparatus will be applied at once, because that joint will have an immediate traumatic reaction to the operative onslaught. The degree of the postoperative reaction, however, will not be nearly so great as it would have been had that joint not had recurrent attacks of synovitis. If the injury to the cartilage had been received quite recently I would have injected the joint with a solution of 2 per cent. formalin in glycerin and produced chemical irritation of the synovial membrane to resist the infection by coffer-damming the tissues. But nature has coffer-dammed well. Dr. Bridge will recall that in former days, when operating in the peritoneal cavity upon a tumor that had been floating around within it for a long time, there was little danger of infection; but that where there had been no previous coffer-damming infiltration, as in the case of a bullet-wound, or when opening an abdomen in which there had been no previous infiltration, fatal peritonitis was more liable to develop.

VISITING PHYSICIAN: How long do you leave the Buck extension apparatus on?

DR. MURPHY: About two weeks.

VISITING PHYSICIAN: Doctor, I want to know how you handled these cases of subperitoneal infection formerly?

DR. MURPHY: We did not handle them—the undertaker usually handled them for us. I shall tell a story of my student-experience in obstetrics. I attended fifteen cases and had seventeen deaths, all the women and two of the children dying: every woman died from sepsis and the two children from tetanus. Within two years after that experience I saw in the same hospital, in the late Dr. John Guerin's service, 203 obstetric cases with but a single death, and that death was due to crushing of a child's skull. What a great contrast! You men of the present day entered medicine after antisepsis and, later, asepsis had come into general use. We, who were burned by the fire of sepsis, appreciate the security obtained by the employment of asepsis and antisepsis much more than those who were not burned.

[*N. B.*—January 5, 1915: Let the record show that now, four months after operation, the result is perfect; that the leg can be flexed to a right angle without causing pain; that the patient uses the expression that he "has had absolutely no pain since the operation"; and that there is no edema and no pain with full flexion. (Consult preceding case.)

FOREIGN BODIES IN KNEE-JOINT—ARTHROTOMY—ABLATION; CHRONIC SYNOVITIS—RESECTION OF SYNOVIAL MEMBRANE

Summary: Clinical history of patient; comments—question of method of approach; exploration of knee-joint *vs.* exploration of abdominal cavity; use of formalin-glycerin injections when not sure of asepsis; effects of foreign-body traumata upon joint; operation—foreign bodies exposed and synovial membrane found congested and thickened from their chronic irritation—semilunar cartilage not loose and left *in situ*—synovial membrane resected; method of suturing synovial membrane; recapitulation; postoperative comments—differentiation from avulsion of tubercles of tibial spine—use of Buck's extension apparatus.

CLINICAL HISTORY OF PATIENT

The patient, a male aged thirty-nine years, was admitted to hospital January 17, 1915. The family and past histories were negative, with the exception that his mother had one leg disarticulated at the ankle-joint and then amputated in the middle third for supposed tuberculosis. She died at the age of sixty-one years of atrophic cirrhosis of the liver.

The present condition is the result of an accident received eighteen years previously—in October, 1897. While playing football the patient's left knee suddenly gave way and he fell to the ground. The knee was manipulated by the attendants, but he was unable to finish the game. He walked from the field limping. He does not know how much motion there was in the knee before the attendants manipulated it; it was, however, very painful and swollen, but there were no abrasions. The knee was bandaged tightly.

Two days after the accident the patient went to school. He walked with a limp and had a dull, aching pain, which lasted five or six days. The swelling did not subside fully until one month after the injury, and during that month he walked with the aid of a cane. At the end of a month he was perfectly well, but there was slight lingering tenderness on pressure on the inner side of the knee.

About one year after the accident, in September, 1898, again while playing football, in turning suddenly the left knee locked, causing the patient to fall and strike the ground with his shoulder. After the fall he was unable to extend or to flex the left knee. After manipulation the locking disappeared, but he was unable to continue the game. Following the disappearance of the locking he walked with a limp, and had pain and swelling in the knee for ten days. He had no chills or fever at this time.

For a period of six or seven years after the second injury the locking recurred two or three times a year. During the past two years the knee has locked upon the least provocation, such as the strain incidental to a misstep from the curbing, or to stepping upon a pebble. When the locking occurs, the foot is inverted and the leg rotated outward. To unlock the knee the patient uses the heel as a fulcrum, grasps the knee and slowly rotates the leg inward until the foot regains its normal position. After unlocking the knee he is able to continue walking. The knee is very painful during this manipulation of unlocking.

During the past eighteen months the patient noticed two or three days after the knee had been locked a pea-sized body on the inner side of the knee-joint: this body is movable and very painful. If, by manipulation, he shifts the little body back into the knee-joint, the pain disappears. The body disappears suddenly, not to reappear until the knee locks again. He has had no chills or fever at any time.

COMMENTS

DR. MURPHY (January 18, 1915): That history is a little difficult to interpret. The question that arises in one's mind is, When external rotation of the foot and leg with locking of the knee occurs, does the little body that appears on the inner side correspond to the displaced semilunar cartilage, or is it a separate, movable fragment? If the semilunar cartilage had really been injured in the first accident, there can be no question about his having injured it again in the second. At present there is a foreign body that disturbs him, but it never appears upon the inner side of the knee until *after* the locking has occurred. Looking at the skiagram you see an additional foreign body situated just one-half inch anterior to the spine of the tibia. If you will look at the other view you will see that the foreign body rests against the spine of the tibia,—that is, against the ledge up to the inner side of the midline,—so that I might have to approach this knee-joint from both sides. I am going to open it first upon the inner side and see if I can find the probably attached foreign body. These foreign bodies are occasionally connected with the ligaments: after they have been present for a time they are torn loose and become floating, and then wander about in the knee-joint. From examining the case I should say that the body always comes back to the same position at the inner side of the midline; that it is probably an attached foreign body with a pedicle, and that it comes in when the knee locks.

The question of the method of attack in this case arises: Shall we employ the central route of approach, splitting the quadriceps tendon, the patella, and the ligamentum patellæ, going directly down through the midline and throwing each split half to each side, or shall we approach the joint from both sides? I should say that I prefer approaching from both sides, because I believe that with this method the patient is incapacitated for a shorter time than when one employs the central route of approach.

For a long time this field of the knee-joint has been hidden completely, except in the presence of grosser lesions. Now, however, exploration of the knee-joint cavity is very much like exploration of the abdominal cavity. When you cannot satisfy yourself clinically as to the nature of the lesion, you can open the joint and explore it just as safely as regards asepsis as when you open and explore the abdomen, provided that you have learned to keep the hands out of the wound. When we began our newer joint-work about fifteen years ago, we prepared a joint for resistance against infection by an injection of a 2 per cent. solution of formalin in glycerin, and this evoked chemical inflammatory reaction in the synovial membrane and thereby developed its resistance to infection, so that after we had operated upon the joint, we found no postoperative effusion poured forth. Unless one be very sure of asepsis, that method of preparation of the joint to resist infection is the procedure which we advise. When one is very sure of asepsis, however, this procedure may be dispensed with and the patient operated upon without such preparation.

As a sequence of recurrent trauma incurred by a foreign body or a movable semilunar cartilage, thickening of the capsule progresses all the time, and finally leads to great impairment of the joint, if not to its destruction. Therefore, when you receive these cases in the very early stages it will be your duty to see that the mechanical source of irritation is removed, whatever that source might be. Before operation I was unable to feel the foreign body that is revealed in the skiagram.

OPERATION

[Applies constrictor to thigh and makes a curved incision.] You see I have made a curved incision down to the level of the tubercle of the tibia and on the inner side of the patella. There is the superficial surface of the fibrous capsule. Here is the intermediate (subsynovial) layer of the capsule, consisting of loose, fatty, vascular and lymphatic tissue, and in it metastases from focal infections lodge. There is the fatty portion of the subpatellar bursa. [Incises the synovial membrane.] Now air has been admitted into the joint. Note the chronic synovitis that has developed in the anterior portion of the synovial membrane. There is one foreign body: here is the other. They are the ones that were shown in the skiagrams. We shall not take out the semilunar cartilage, as it appears to be well anchored in place. We shall, however, ablate the foreign bodies and resect the thickened anterior portion of the synovial membrane.

Let the record show that upon opening the joint the anterior portion of the synovial membrane was found congested and thickened as the sequence of the chronic irritation from the foreign body.

[Stitches capsule.] Now I am inserting this stitch outside of the synovial membrane, catching the inner portion of the fibrous capsule, so as to make an ectropion of the cut edges. There is the margin of the synovial endothelial lining, and the suture is placed superficial to that line, and therefore outside of the joint.

RECAPITULATION

Let the record show that after exposing the cavity of the knee-joint two foreign bodies were found close to the midline and a little to the left of the center of the patellar ligament; that these bodies were entirely detached and readily removed, and the joint was closed; that next the elongated, anterior portion of the synovial membrane was resected because of its swollen, edematous, and thickened condition, and the fear of its becoming subsequently pinched between the surfaces of the joint and traumatized.

[Removes constrictor from thigh.] There is no bleeding at all after the constrictor has been removed. I am uniting the fibrous capsule, thereby obliterating the dead space. [Closes wound in skin.]

Let the record show that all this work was executed without contacting of the wound with the gloved hand or with that part of any instrument that had touched the gloved hand.

POSTOPERATIVE COMMENTS

The skiagram was of much value in locating these foreign bodies and in facilitating their exposure. But for the skiagram one would have suspected the presence of the condition described by Corner, of London, namely, avulsion of the tubercles of the tibial spine through the crucial ligaments, and that it was the avulsed tubercles with their crucial ligaments that were causing the foreign-body trouble. The skiagram revealed the foreign bodies upon the *left* side, although I said they were upon the right. When they started to cause the patient trouble they continued troubling him until he could displace them to the right, that is, to the outer side (toward the center of the joint).

The Buck extension apparatus has been applied already. We prepare this apparatus before bringing our patients into the operating room, so as to prevent joint-contacting during the process of repair. We apply Buck's extension in all these cases because it aids the process of repair by affording physiologic rest and opposing harmful muscular contraction.

[*Note.*—There was primary union with an uninterrupted and complete recovery.]

SARCOMA OF POPLITEAL SPACE—ABLATION OF TUMOR

Summary: Clinical history of patient; comments—a neoplasm should be removed when small; amputation of limb *vs.* local ablation of neoplasm; difficulties anticipated in removing tumor from popliteal space; pathologic aspects of neoplasms—division and subdivision of sarcomata and other tumors; operation; recapitulation; postoperative comments.

CLINICAL HISTORY OF PATIENT

The patient, a female aged sixteen years, was admitted to hospital September 6, 1915. The family history is negative. In 1910 she was operated upon for club-foot.

Over two years previous to admission, in July, 1913, while consulting a physician regarding her right-sided club-foot, her attention was called to the presence of a hard mass about the size of a walnut on the calf of the right leg. There were no pain and no tenderness, and the surface of the calf was not much elevated by the tumor, which was movable and not attached to the skin.

The tumor gradually increased in size until four months previous to admission, when, for the first time, the patient noticed occasionally an ache in the leg after exertion and that the tumor was spreading around the circumference of the leg. Two months later there began sharp, stinging pains in the region of the tumor, especially in the morning on arising, or after sitting in one position for a long time. During the last two months the tumor has increased rapidly in size; it is hard.

One week previous to admission, on August 31, 1915, an incision was made through the skin, exposing the tumor, and a piece of the latter was removed for microscopic study.

COMMENTS

Dr. Murphy (September 11, 1915): Two years ago a mass as large as a walnut was noticed on the calf of the leg. A mass so large as that has no license to be on the calf of the leg, and the time to ablate it is when it is the size of a walnut, and preferably when smaller. You do not want to let *any* neoplasm grow until it becomes so large that you must have a derrick to lift it up with. I want to emphasize the dangers and the evil sequelæ of procrastination.

In this patient's case, while the neoplasm was still comparatively small, a very able surgeon removed a piece of the tumor for microscopic study, and it proved to be a fibromyxosarcoma of the spindle-cell type, springing from the deep fascia. The microscopic finding was corroborated by two pathologists. The first one, young in experience, thought that a hip-joint amputation was indicated. The second, older in experience, said that, so long as sarcomata of this type are often local tumors which do not recur when removed, and are, therefore,—shall we say?—benign, an effort should be made to ablate the neoplasm alone, and we are happy to act upon his suggestion.

The first recognition of this tumor was a positive announcement to the physician that some action should be taken: if it were an innocent tumor,—one even so innocent as a lipoma,—there would be nothing gained by waiting, and it should be ablated. The skiagram shows the tumor invading the popliteal space and involving the popliteal vessels apparently.

In an attempt to remove the tumor locally I shall have a host of difficulties to contend with. First, in all probability I shall have hard work in releasing from the tumor the internal and the external popliteal nerves; secondly, if we find it necessary to ligate the popliteal artery, there is the possibility of gangrene of the foot arising, but it is a question between that source of danger and primary amputation at the hip-joint. If the patient were your own daughter, you would first try to ablate the tumor.

Taking up the pathologic aspects of these neoplasms, we have learned by recent experiments that many sarcomata may be removed together with their capsule, and a permanent cure effected. This has been particularly true of the non-metastasizing giant-celled type of bone-sarcoma, which is being removed more and more frequently by resection of the portion of the shaft involved in the tumor, and restoration of the shaft to its original length by the intramedullary insertion of a tibial transplant.

By dividing the sarcomata first into those of the simple connective tissue (Mesoblastic—Class A) type, and then subdividing the mesoblastic type into those originating from—(1) The endothelial cells of the lymph-spaces; (2) the endothelial cells of the blood-spaces; and (3) the endothelial cells of the meninges, you have them fairly well classified, and you may keep them clearly in mind by this scheme of subdivision. The second division of sarcomata is into those of the special lymphoid tissue type, mentioned under Class B. The third division includes the melanoblastoma tumors, which are mentioned under Class D, and the subdivisions under this class are those with dark pigment, known as the melanotic type, which type includes, in addition, the melanotic adenomata and also the melanotic carcinomata. Finally, there is the non-sarcomatous group of tumors known as the epithelio-blastomata, the subdivisions of which are papilloma, adenoma, and carcinoma. By keeping this classification in mind, you will have a fairly good working knowledge of the various types of tumors.

OPERATION

[Constrictor applied to thigh and tightened until arterial pulsations below were lost.] I am going to make the first part of the incision in the transverse direction, so as to avoid dividing the crease of the popliteal space, which is one of the sites where keloids and painful scars are wont to develop. I am excising the scar that resulted from the previous operation. . . . Next, I shall enter the upper portion of the popliteal space and expose the sciatic nerve at its bifurcation. Then I shall follow the external and internal popliteal nerves down to determine where these divisions of the sciatic nerve pass to, as I may be able to preserve them. . . . There is the internal popliteal nerve: it is pushed far to the outer side by the tumor, and over against the external popliteal nerve (Fig. 334). The natural planes of cleavage are disturbed here as a result of the previous operation. . . . There is the outer head of the gastrocnemius

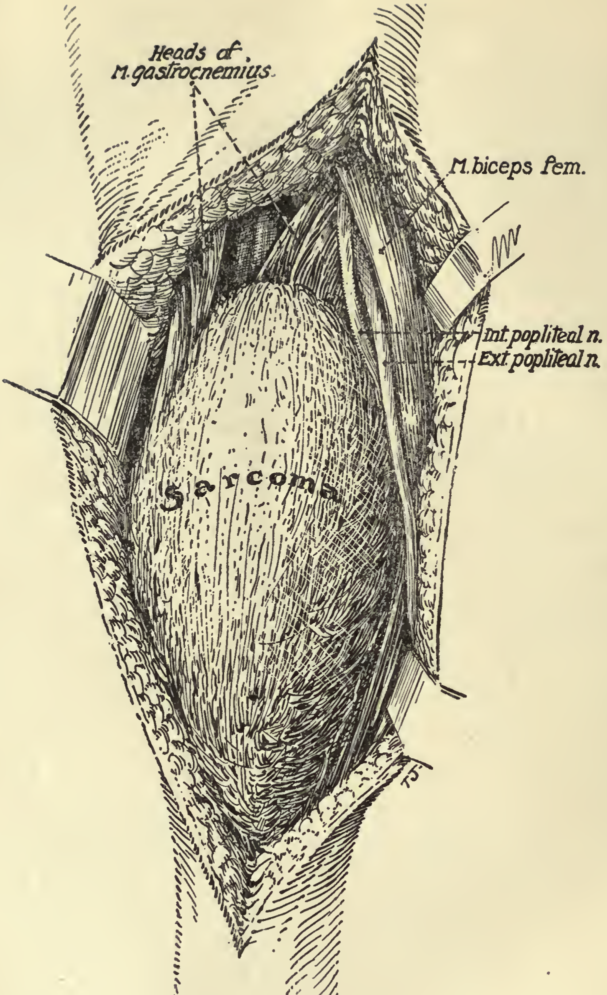

Fig. 334.—Sketch showing development of operative field and exposure of sarcoma preparatory to ablation. Note orientation of sarcoma to structures of popliteal space, the internal popliteal nerve being displaced by sarcoma to outer side, over against the external popliteal nerve, while the popliteal vessels are exposed in upper angle of wound. Both heads of gastrocnemius muscle are invaded by growth. Sketch made at operating-table.

muscle; it is invaded by the tumor. Following the external popliteal nerve downward we reach the head of the fibula.

Now I have developed the operative field on both sides, and I have exposed it fairly well from the inner side. . . . There is the posterior surface (ham) of the femur, with a portion of the joint-capsule showing. It requires very much care in this situation in ablating this tumor, but I am gradually getting my bearings. . . . There is the internal popliteal nerve, displaced to the outer side by the bulk of the neoplasm. Here is the inner head of the gastrocnemius in the field: the tumor seems to have originated in the popliteal fascia covering this head. . . . This tissue is infiltrated from the previous operation. Judging from the gross appearance of the neoplasm the diagnosis of sarcoma appears to be correct. It seems to be one of those fibrous growths—a fibrosarcoma. . . . I am excising both heads of the gastrocnemius muscle, together with the overlying fascia and the underlying periosteum. The constrictor is now removed, and there is surprisingly little bleeding in proportion to the extent of the dissection. Now we shall tie these vessels, whose open ends are revealed by the blood released upon removal of the constrictor. [Rubber drainage-tube inserted and incision closed.]

RECAPITULATION

Let the record show that the tumor originated in the popliteal fascia covering the inner head of the gastrocnemius muscle, invaded that head, and extended over into the outer head of that muscle, but not quite so far as its attachment to the posterior surface (ham) of the femur; that the tumor projected three inches beyond the level of the skin; that after incising the skin, we first identified and isolated the internal popliteal nerve, which was displaced to the outer side, and then identified and isolated the external popliteal nerve upon the outer side, exposing and removing both heads of the gastrocnemius, including three inches of adjacent femoral periosteum; that the soleus muscle was not removed, because not involved in the neoplasm; that there was no evidence of metastases; and finally that a rubber drainage-tube was inserted, and was brought out through both angles of the wound.

POSTOPERATIVE COMMENTS

I am daubing the operative field with 5 per cent. phenol, because there has been such an extensive handling of the tissues. This drug, in addition to its germicidal action, has a good hemostatic effect upon the small blood-vessels. We have reconstructed the popliteal space and have adjusted the tissues compactly, so as to coffer-dam dead tissue-spaces.

[*N. B.*—September 13, 1915: The drainage-tube is removed to-day, forty-eight hours after the operation.]

[*Postscript.*—The following note was received recently from the patient operated upon for empyema by the Estlander method, whose case was published in the CLINICS for October, 1915, pp. 885–896:

"On November 1, 1914, I discontinued the use of the vaccines, since I found that I was getting no further reactions from them. One month later, on December 1, 1914, I resumed my work in the lumber yard. The wound has not reopened since I removed the drainage-tube, and I am feeling fine."]

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